Special Bulletin No. 198
1946

SILICOSIS

AND ITS PREVENTION

By
Adelaide Ross Smith

New York State Dept. of Labor
EDWARD CORSI
Industrial Commissioner

Special Bulletin No. 198
1946

SILICOSIS

AND ITS PREVENTION

By
Adelaide Ross Smith

New York State Dept. of Labor
EDWARD CORSI
Industrial Commissioner

Division of Industrial Hygiene and Safety Standards
EDWARD A. NYEGAARD
Deputy Industrial Commissioner
LEONARD GREENBURG, M.D., Exec. Director

State of New York

DEPARTMENT OF LABOR

Albany Office
The Alfred E. Smith
State Office Building

New York Office
80 Centre St.

EDWARD CORSI
Industrial Commissioner

ABRAHAM A. GOODMAN
LOIS B. HUNTER
ESTHER DORN LONGSTREET
EDWARD A. NYEGAARD
Deputy Industrial Commissioners

BERTHA J. DIGGS
Secretary

DR. LEONARD GREENBURG, M.D.
Executive Director

DIVISION OF INDUSTRIAL HYGIENE AND SAFETY STANDARDS

INTRODUCTION

This bulletin, the first edition of which appeared in 1938, was compiled in order to furnish a convenient reference manual for those interested in silicosis and allied pulmonary conditions.

It is revised to include recent material and republished at this time to meet a continuing demand.

ADELAIDE ROSS SMITH, M.D.

ILLUSTRATIONS

TABLE OF CONTENTS

Granite Surfacing--With and Without Dust Control

CHAPTER I

SIGNIFICANCE OF SILICOSIS

Pneumoconiosis

The word pneumoconiosis is a general term signifying any pathological condition of the lung due to the inhalation of dust. Qualifying terms such as anthracosis, asbestosis and silicosis are used to indicate the type of dust involved—as coal, asbestos and silica. Mineral dusts containing silica in the free or combined state, especially the former, far outrank all other types of dust in the causation of disabling pneumoconiosis.

Definition of Silicosis

Silicosis is defined by the American Public Health Association as follows:

"Silicosis is a disease due to breathing air containing silica (SiO_2) characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present) and by characteristic X-ray findings." It is more or less arbitrarily divided into three stages.

Public Health Aspect of Silicosis

Before describing the properties of silica itself and the pathology of the disease it will perhaps give a better understanding of its importance to consider it from the public health aspect in terms of the number exposed to the possible hazard of contracting it and the effect of exposure upon mortality statistics.

Number Exposed to Silica Dust in United States and New York State

Lanza and Vane[2] estimate that there are approximately 450,000 workers exposed to silica dust in the chief mining, quarrying and manufacturing industries of the country with a good many more thousand previously exposed but now engaged in other work. This is probably a conservative estimate.

In New York State the estimate of the number of people exposed to definitely injurious dust is tentatively put at 7,500; with a middle group of between 13,000 and 14,000 exposed to dust of a moderately harmful type, from the aspect of both concentration and nature; and a third group of about 35,000 exposed to a questionable dust hazard, the extent of which cannot at the moment be appraised.

Silicosis and Tuberculosis

All analyses of mortality statistics among those exposed to silica point to one thing: The increased frequency of deaths from tuberculosis among them.

This practically unvarying association with tuberculosis constitutes by far the most serious feature of the disease; one which has been recognized for hundreds of years. Modern statistical evidence of the association is abundant.

England and Wales

The following table presents the standardized respiratory tuberculosis mortality rates for males between ages 20–65 for the years 1930–1932 for England and Wales from the report of the Registrar General.[3]

Occupation	*Respiratory Tuberculosis Mortality Rates* *Per 100 Standard*	*Per 100,000*
All males	100	124
Tin and copper mine workers below ground	867	1,370
Cutlery grinders	757	950
Metal grinders	275	340
Slate miners and quarriers	256	317
Sandstone miners and quarriers	179	222
Stevedores	239	295
Potters	233	288
Barmen	212	262

In this table are presented the eight occupations having the highest mortality rates from tuberculosis as compared with the rate for all occupied and retired males. Of the eight occupations listed, six are associated with the inhalation of quartz dust. It will be observed that tin and copper underground miners have a tuberculosis rate more than ten times that for all occupied or retired males.

Life Insurance Statistics

In a study made by 12 life insurance companies in this country in 1928 quoted by Lanza and Vane,[2] "the occupations showing the highest ratios of actual to expected deaths from tuberculosis were, in order: Underground lead and zinc miners, granite and sandstone cutters, copper miners and gold and silver miners.

"All of these workers were exposed to a serious silica hazard. The ratio of actual to expected deaths from tuberculosis were respectively 1,833 per cent, 976 per cent, 913 per cent and 804 per cent. There were actually 60 deaths from tuberculosis among these men as against six expected. Tuberculosis was responsible for one-half of all the deaths of lead and zinc miners, 29 per cent of the deaths of copper miners, and 20 per cent of those among gold and silver miners. In all these three mining classes, deaths from

tuberculosis exceeded deaths from accidental injuries. Among iron miners who are, on the whole, probably less exposed to silica dust than are the other mining classes mentioned, we find that the ratio of actual to expected tuberculosis deaths is only 260 per cent. There were 16 deaths from tuberculosis among granite cutters as against 1.7 expected."

Anthracite Miners

In a study of anthraco-silicosis among hard coal miners made by the United States Public Health Service,[6] the control group, not exposed to dust, was found to have a tuberculosis rate of less than one per cent, whereas among those with early and advanced anthraco-silicosis it was 15.3 and 43 per cent respectively.

Ax-Grinders

Winslow and Greenburg[7] quote an interesting study of tuberculosis in an ax-grinding plant in Connecticut made by Drury which covered the death certificates of the workers for the previous 20 years. Sandstone wheels with a high percentage of quartz had been used. The rate among grinders and polishers was 1,900 per 100,000. Among other persons in the mill it was 160, and for the State of Connecticut as a whole 150 per 100,000.

Granite Workers

In the U. S. Public Health Service study of granite workers it was found that tuberculosis was recorded as the cause in 65 per cent of 631 deaths among cutters, occurring between 1900 and 1925. During the period of the study the death rate for tuberculosis was 14 per 1,000 granite workers, a rate, as the authors point out, in excess of the death rate for all causes in the general population.[5]

Post-Mortem Studies

Dr. Irvine, in the 1929 report of the Miners Pthisis Medical Bureau, says, "A measure of the extent of the predisposition to tuberculosis in cases of silicosis is afforded by the fact that of all deaths that had occurred (among 1,623 silicotics) some 75 per cent have been due directly to tuberculosis."

In Great Britain 59.4 per cent of autopsies on silicotic subjects revealed coexistent tuberculosis and Gardner reports that of 167 autopsy specimens that have come to the Saranac Laboratory with a history of industrial exposure to various dusts, the incidence of tuberculosis has been 65.2 per cent in cases with silicotic nodulation.

Silica Exposure and High Mortality from Other Causes

It seems possible in view of the statistical analysis made by Collis and Yule[8] that the presence of silica in the body in undue amounts not only predisposes to pulmonary tuberculosis but has an unfavorable effect on other organs. Comparing mortality statistics in a large group of industrial workers exposed to silica dust with another industrial group not so exposed and with the general population, they found definitely increased rates for diseases of the heart, gastro-intestinal tract and kidneys among the former. So striking was the difference that they declare silica to be a general body poison as is lead. Gardner[9], however, does not find such a conclusion justified by his experimental studies.

CHAPTER II

CAUSE OF SILICOSIS

Chemical Properties of Silica

Silica has the chemical formula SiO_2 indicating that it is a combination of the element silicon (Si) and oxygen (O). It occurs by itself in the "free" form, and as such, is variously known as silicon dioxide, or "free" silica. In combination with other elements, it is known as "combined silica," but it is free silica which concerns us especially because in this form it causes more damage to the lungs than when it is in the combined state.

Structure

Free silica, silicon dioxide, may be either crystalline or non-crystalline (amorphous) in structure. Crystalline silica, which is simply quartz, is a white or colorless, extremely hard substance with a specific gravity of 2.66. When heated for a long time at about 1,000° C. it forms a second variety known as tridymite with a specific gravity of 2.33. A third variety, cristobalite, specific gravity 2.34, occurs when quartz is heated for periods not long enough to form tridymite. Non-crystalline (amorphous) silica occurs as a fine white powder.

Thermal Properties

Silica assumes the vitreous, molten form more easily than any other mineral substance, melting to colorless quartz glass in the oxyhydrogen blowpipe beginning at about 1,600° C. The coefficient of thermal expansion of quartz glass is very small, so that it possesses in a high degree the property of being able to withstand rapid cooling without cracking.

Silica is reduced by carbon in the electric furnace to form silicon carbide, and by magnesium to amorphous silicon.

Solubility

Crystalline and vitreous silica are insoluble in water and in all acids except hydrofluoric, but they are slowly soluble in aqueous solutions of alkaline hydroxides and carbonates. Fused silica is readily soluble in phosphoric acid and the alkalies.

Silica is chemically inactive at ordinary temperatures but at high ones acts as an acid anhydride and combines with the bases and many metallic oxides to form silicates. When soluble alkaline silicates are treated with acids, silicic acid, an amorphous, gelatinous substance is obtained. This is soluble in water and acids and is readily dissolved by dilute solutions of alkali hydroxides and carbonates.

Occurrence of Silica in Nature

Silica is one of the commonest minerals. In one form or another it enters into 60 per cent of the earth's crust and forms whole mountain ranges in certain countries.

Free Silica

Examples of free silica in the pure crystalline form are quartz and rock crystal but there are many other minerals whose essential composition is quartz but which have been changed in color or form by the addition of small amounts of other substances, usually oxides of other elements. Many of these are used chiefly for ornaments and jewelry but some, such as buhrstone and flint, have important industrial uses ts well. A list of these quartz rocks includes the following:

Agate	Cairngorm	Needle stone
Adventurine	Chrysoprase	Onyx
Amethyst	Certine	Opal
Bloodstone	Flint	Plasma
Brazilian pebble	Hairstone	Phrase
Buhrstone	Hyalite	Rose quartz
Carnelian	Jasper	Sard
Cat's eye	Jaspilite	Smoky quartz
Chalcedony	Lydian stone	Tiger's eye
Chert	Milk quartz	

Common rocks containing a high proportion of free silica are:

Sand-stone, essentially grains of quartz with some feldspar, mica and other materials added.
Quartzite, a product of sandstone.
Ganister, a sedimentary rock with a very high percentage (about 98 per cent) free silica.
Granite, composed essentially of quartz and feldspar, containing 25 to 35 per cent of free silica.
Pegmatite, a form of granite.
Ordinary sand is made up almost entirely of grains of quartz.

Amorphous (non-crystalline free) silica forms the composition of opal and is associated with quartz in certain other stones such as chert, flint and chalcedony. It is chiefly represented by diatomaceous earth and tripoli or "rotten stone."

Diatomaceous earth is a soft earthy rock composed of the skeletons of small aquatic plants, resembling chalk in appearance. It is capable of holding four times its weight of water and is a poor conductor of heat, sound and electricity. Tripoli is a porous rock which results from the natural decomposition of sandstone.

Silicates

The silicates, combinations of silica with other minerals, enter into the composition of every rock species and form the essential substance of such common materials as clay, mica, feldspar and slate.

Certain silicate minerals of fibrous structure, such as asbestos (hydrous magnesium silicate) and tremolite talc, give rise to forms of pneumoconiosis differing in several important respects from silicosis. These will be described later.

The non-fibrous silicates are not found experimentally to cause progressive tissue changes, but under conditions of industrial exposure pneumoconiosis from several, notably mica and pumice has been reported. It seems probable that when "silicatosis" appears it is due to the incidental presence of small amounts of free silica which are effective because of high dust concentrations.

Industrial Uses of Silica

Silica has many properties which give it industrial value as for example its hardness, resistance to acid and to quick temperature change, its crystalline and decorative properties and, in the amorphous form, its value as a filtering medium and non-conductor.

It is used in a variety of different forms, i.e., in the massive form, both roughly broken and cut to different sizes and shapes; in the original condition as pebbles or as grains; crushed to a coarsely granular form or ground to a fine powder.

Its chief uses and corresponding states have been tabulated by Ladoo[15] as follows:

Uses of Silica	Types of Silica Used
Abrasive uses:	
In scouring and polishing soaps and powders.	Quartz, quartzite, flint, chert, sandstone, sand, tripoli, and diatomaceous earth; all in finely ground state.
In sandpaper	Quartz, quartzite, flint, sandstone and sand; coarsely ground and closely sized.
In sand-blast work	Quartz, quartzite, sandstone and sand, crushed into sharp angular grains uniform in size.
Metal buffing, burnishing and polishing	Ground tripoli and other forms of ground silica.
For sawing and polishing marble, granite, etc.	Sharp, clean sand graded into various sizes.
As whetstones, grindstones, buhrstones, pulpstones, oilstones.	Massive sandstone from very fine to moderately coarse grained.
Tube-mill lining	Chert, flint, and quartzite in dense, solid blocks.
Lithographers' graining sand	Medium to fine sand or rather coarsely ground silica and tripoli.
Tube-mill grinding pebbles	Rounded flint pebbles.
In tooth powders and paste	Various forms of pure silica finely ground.
Wood polishing and finishing	All forms of silica ground to medium fineness.
Refractory uses: In making silica fire brick and other refractories.	Fairly pure quartzite known as ganister: not less than 97 per cent. SiO_2 nor more than 0.40 per cent alkalis. Tightly interlocking grains desired.
Metallurgical uses:	
In making silicon, ferro-silicon, and silicon alloys of other metals, such as copper.	Moderately pure sand, massive crystalline quartz, sandstone, quartzite, or chert.
As a flux in smelting basic ores	Massaive quartz and quartzite.
Foundry-mold wash	Ground sandstone, quartz, and tripoli.

Uses of Silica	Types of Silica Used
Foundry parting sand	Fine sand and ground tripoli.
Chemical industries:	
As a lining for acid towers	Massive quartz or quartzite.
As a filtering medium	Massive diatomaceous earth and tripoli, sand finely granular quartz or quartzite, finely ground tripoli, diatomaceous earth, and other forms of silica.
In the manufacture of sodium silicate	Pure pulverized quartz sand, pure tripoli, and diatomaceous earth.
In the manufacture of silicon carbide	Pure quartz sand.
Paint: As an inert extender	Finely ground crystalline quartz, quartzite and flint; also finely ground sandstone, sand, and tripoli.
Mineral fillers: As a wood filler	Finely ground crystalline quartz, quartzite, flint tripoli, and other types of ground silica.
In fertilizers	As above.
In insecticides	As above.
As a filler in rubber, hard rubber pressed and molded goods, phonograph records, etc.	Finely ground silica of all types.
In road asphalt surfacing mixtures	As above.
Ceramic uses: In the pottery industry as an ingredient of bodies and glazes.	Flint, tripoli, and chert, amorphous silica preferred: also all other forms of very pure silica, all finely ground.
Building stone	Cut granite and sandstone.
Monuments, paving blocks	Cut granite.
In the manufacture of ordinary glass	Pure quartz sand.
In the manufacture of fused-quartz chemical apparatus, such as tubes, crucibles, and dishes.	Very pure massive quartz preferred.
Decorative materials: In the manufacture of gems, crystal balls, table tops, vases, statues, etc.	Rock crystal, amethyst, rose quartz, citrine quartz, smoky quartz, chrysoprase, agate, chalcedony, opal, onyx, sardonyx, jasper, etc.
Insulation:	
Heat insulation for pipes, boilers, furnaces, kilns, etc.	Massive and ground diatomaceous earth.
Sound insulation in walls, between floors, etc.	As above.
Structural materials: Sandline brick	Moderately pure, sharp, angular sand, preferably finer than 20-mesh, together with a small percentage of finely pulverized silica.
Optical quartz: For the manufacture of lenses and accessories for optical apparatus.	Clear, colorless, flawless rock crystal or massive crystallized quartz.
Preservation of stone	Organic silicon compounds.

Silicosis-Hazardous Industries

It is apparent that exposure to silica dust in industry will occur not only (1) in the **use** of siliceous materials as listed above but also (2) **in obtaining or preparing siliceous materials,** such as in:

> Quarrying sandstone, granite, slate or mica; milling sand, flint or other silica-containing substances; cutting and surfacing granite or sandstone;

and (3) **in any dust-creating operation where contact with siliceous material is unavoidable,** as in mining, excavating and tunnelling in hard rock, or where silica dust is present incidentally.

In modern times silicosis was first recognized when a Commission was appointed in 1902 to study conditions in the gold mines on the Rand in South Africa. Since then investigations all over the world have brought to light its very widespread occurrence. The results of many of these investigations were summarized in the Report of the International Conference on Silicosis held at Johannesburg in 1930.[16] The following table shows the industries in which it has been most commonly reported:

Industry	Source of silica exposure	Operations offering exposure
Abrasives: scouring powders, soaps.	Ground sand	Grinding, mixing, packing.
Excavating	Rock dust	Drilling, blasting, hauling.
Foundries	Sand used in molds and in sand blasting. Parting compounds.	Sand blasting, cleaning, sand conditioning.
Glass	Sand is a constituent of glass; also used in sand blasting.	Mixing, polishing, sand blasting.
Metal hand tools, instruments, etc.	Dust from grindstone when natural stones are used.	Grinding, also dressing surface of stones.
Milling sand, flint, slate	Dust from material milled	Milling, packing, conveying.
Mining (hard rock)	Rock dust	Drilling, blasting, hauling.
Pottery: including tiles, electrical fittings.	Dust from ingredients; china-stone, flint and feldspar.	Mixing, making, mould making, biscuit placing and brushing, fettling, etc.
Quarrying granite, sandstone, etc.	Dust from stone quarried	Drilling, blasting, cutting, hauling, etc.
Refractory materials, fire brick, etc.	Dust from materials used: ganister, quartzite, sandstone.	Crushing, grinding, mixing, and subsequent manipulation of dried bricks, etc.
Sandblasting: buildings, railroad cars, etc.	Sand used to clean or remove paint, etc.	Sandblasting.
Stone works: granite, sandstone.	Dust from stone	Cutting, surfacing, sandblasting.
Tunnel construction (hard rock)	Rock dust	Drilling, blasting, hauling.
Vitreous enamelling of sanitary ware.	Silica in enamel	Mixing, spray-coating.

Isolated cases have also been reported among such workers as grave diggers, leather dressers and metallurgists.[17]

CHAPTER III

CHARACTERISTICS OF SILICOSIS

Factors in the Development of Silicosis

At this point the question naturally arises: "What exact conditions of exposure to silica dust determine the development of silicosis?"

Unfortunately, the conditions influencing its development are so numerous and variable that a simple answer is impossible. The following all play a part, the chief factors being silica content and character, length and intensity of exposure, to which must be added individual susceptibility:

Type of Silica in Dust

By definition, silicosis comes from breathing air containing SiO_2 or free silica which, in the crystalline or quartz form, is most injurious. Amorphous (non-crystalline) silica is capable of producing a less severe degree of silicosis but exposure to this form is limited. The silicates (silica in the combined form) also are, as stated above, with the exception of asbestos, much less harmful than free silica.

Per cent of Free Silica in Dust

Since silicosis comes from breathing free silica, obviously one of the factors in promoting its development in any industry will be the amount or percentage of free silica in the dust under consideration. Dusts with higher percentages of free silica are more dangerous under similar conditions than those with less.

Thus the dust produced in grinding or pulverizing sand for use in abrasives is more dangerous than the dust in a cement plant because pure sand is composed of approximately 99 per cent free silica and cement of only six to eight per cent.

Size of Dust Particles

The smaller the size of particles in any dust, the longer they will stay in suspension in the air, subject to being breathed and, consequently, the more harmful the dust. Particles above 10 microns in size settle with relative rapidity. A micron is approximately one twenty-five thousandth of an inch.

The size of the particles in dusts of various kinds differs of course depending on the material, but in many processes involving exposure to silica dust the particles tend to be extremely small.

The latest data indicate that the majority of particles in an industrial dust and in silicotic lungs are less than one micron in size.[18] This means of course that much of the dust in such operations is invisible in the ordinary microscope. Such dust is so fine that it can be thought of rather as a gas than as a dust. After all visible dust has been removed from an operation, the invisible dust present may still be sufficient to cause silicosis.

Length of Exposure to Dust

Then there is the factor of duration of exposure. The longer the exposure, the greater the risk. Intermittent dust exposure, providing it is not excessive, is less likely to give rise to silicosis than work where contact with dust is continuous. This is probably not true, however, of intermittent high exposures.

Degree of Dustiness

Of great importance is the degree of dustiness or the number of particles in a given amount of air. Naturally the more dust of a harmful character present, the greater will be the liability to silicosis.

Dust is estimated in this country as the number of particles per cubic foot of air. Dust counts in jobs of moderate dustiness range between 20 and 50 million particles per cubic foot, while in intensely dusty work the count may reach a billion particles or more.

Since the harmfulness of a dust depends not only on the number of small particles and percentage of free silica but also on the other factors mentioned, particularly length of exposure, it is extremely difficult to say what constitutes a "safe" dust count under these varying conditions.

Standards of Permissible Dustiness

In the granite industry where the granite contains 35 per cent of free silica, a count of 10 million particles per cubic foot is apparently safe. Workers exposed to dust giving counts of that amount or less have shown no increase of respiratory disease. Presumably with dust containing 70 per cent free silica a count half as high would be required for safety, but definite standards must await future investigation and experience.

The study of anthraco-silicosis made by the U. S. Public Health Service[6] showed that employment in an atmosphere containing less than 50 million dust particles per cubic foot would produce a negligible number of cases of anthraco-silicosis when the quartz content of the dust was less than five per cent.

Where the quartz content was about 13 per cent, a safe limit appeared to be 10 to 15 million particles per cubic foot. The limit of toleration for rock workers exposed to dust with about 35 per cent quartz was set tentatively at five to 10 million particles per cubic foot of air.

Cummings, quoted by Drinker and Hatch, from a consideration of general experience, suggests five million particles per cubic foot as a threshold for dust high in quartz.[18]

In the National Silicosis Conference Report[18a] the following statement is made in regard to "safe" dust concentration:

> "In general, dust concentrations of less than five million particles per cubic foot of air are considered safe even in cases where the dust contains a high percentage of free silica. To obtain a more definite correlation of these two factors it has been suggested that the dust count be multiplied by the percentage concentration; then if the result is under five million, one can be almost sure that the conditions are safe. But unfortunately the reverse is not so definite; if the result is more than five million, one cannot say with assurance that the conditions are unsafe."

In New York State, Codes (see page 65) for dust control in rock drilling, stone crushing and stone cutting and finishing require that when the rock involved contains more than 10 per cent of free silica, dust counts shall not be permitted to exceed 10 million particles per cubic foot. When rock contains 10 per cent or less of free silica, counts may not exceed 100 million particles per cubic foot. The Stone Cutting and Finishing Code contains a further provision that if free silica is present to the extent of 70 per cent or more dust counts shall not exceed 5,000,000 particles per cubic foot.

Individual Susceptibility

It is apparent with silicosis as with most diseases, that there is a great difference in individual susceptibility but the chief reasons for this difference are obscure. In some cases early respiratory infection may be a predisposing factor. Relative inefficiency of the nose in filtering dust may be another. The rate of respiration and the type of breathing of a given individual has also been suggested as a possible factor in individual susceptibility.

It is held that there is a general racial susceptibility among negroes to silicosis as there is to tuberculosis. A recent investigation along these lines among 4,066 foundry workers by Greenburg, Siegal and Smith[85] is of especial interest in this connection because the silicosis rates found among the 180 colored foundry workers included in this study were lower than those for the group as a whole (1.7 per cent as compared with 2.7 per cent). Several factors, however, appeared to play a part in these low rates: (1) the colored group, as a whole, was somewhat younger than the entire group—55 per cent being less than 40 years of age compared with 42 per cent of all foundry workers in this age group; (2) their duration of foundry exposure was less—86 per cent having been exposed less than 20 years compared with 60 per cent of the entire group; (3) a larger proportion of them were employed in foundries of the "combined" type—55 per cent as compared with 34 per cent of the entire group—wherein the silicosis rate was found to be the lowest of the four types of foundries. The distribution of the colored workers by specific occupation followed in general the distribution of the white workers examined.

Length of Time Necessary to Develop Silicosis

Silicosis may develop in several months under continuous intense exposure to fine dust of a very high quartz content; but it usually takes many years if there is intermittent exposure to a slightly dusty process where the material is coarse and low in quartz content, and the individual not particularly susceptible.

Action of Silica on Body Tissues—Experimental Studies

Toxicity

Silica is a definite tissue poison. Gardner,[14] Gye and Purdy[19] and others have found that when a suspension of minute silica particles is injected into the tissues, an acute inflammatory reaction occurs which is followed by necrosis or destruction of cells. This in turn is followed by a healing process characterized by the formation of a nodule of connective tissue fibres. Other dusts such as silicon carbide, emery, diamond, coal and iron may cause an inflammatory reaction but without necrosis or nodule formation.

As a Colloid

Heffernan and Green[22] suggested that the action of silica on animal tissues does not depend on toxic properties but rather upon its properties as a powerful colloid, absorbing body fluids and otherwise interfering with normal processes.

Cummins and Weatherall[23] found that silica sol interfered with the bactericidal action of the blood against bacillus typhosis, though no effect was shown on the growth of tubercle bacilli.

Solubility

Silica is apparently soluble in body tissues. Mills[20] reports that when particles of the fresh water sponge, which is similar in composition to quartz, are introduced into the tissues of animals they are slowly but definitely dissolved. Belt[21] suggests that when silica dust is inhaled it gradually changes in the lungs from the crystalline form to silica sol and eventually to silica gel. In the intermediate stage it injures lung tissue producing the disease silicosis, and reduces its resistance. This action continues and the disease progresses until all the toxic silica sol is changed to the inert gel.

Support for this idea is furnished by the experiment reported by Mills in which, after particles of fresh water sponge were placed in the lung tissue of a dog, definite fibrosis was found, suggesting a concomitant injury developing as the sponge dissolved. The production of fibrosis, over-development of connective tissue, in the lung is the characteristic feature of silicosis.

The belief that the irritating properties of silica depend on its solubility in body tissues appears to be substantiated by the work of Denny, Robson and Irwin (see also page 64) who showed experimentally that when silica particles were rendered insoluble by a coating of aluminum hydrate silicosis could be prevented.

Effect on Tubercle Bacilli

Experiments, interesting in the light of the clinical association of silicosis with tuberculosis, have been performed by numerous investigators. Gye and Kettle[24] found that when they injected silica and tubercle bacilli together, the bacilli were apparently protected by some charactertistic of the silica abscess and multiplied rapidly. In animal experiments, if a silica abscess were produced in one groin and an abscess from another irritating substance in the other groin, tubercle bacilli injected subsequently by vein showed a tendency to concentrate in the silica abscess.

Gardner,[25] working with guinea pigs, found that partially healed areas of tuberculous infection were reactivated and made to progress by the inhalation of quartz dust.

These findings were confirmed by Dowd,[26] who concluded from his work, also with guinea pigs, that inhaled silica dust alters tissues so that they become more favorable to the growth of the tubercle bacilli. Vorwald and Delahant[26a] threw some light on the nature of this alteration. They found that silica inhibits the development of acquired resistance to the tubercle bacillus.

Behavior of Silica in the Body

In spite of being a tissue poison in concentrated doses, silica in small amounts is a natural constituent of all animal tissue and is present in certain foods.

Urinary Excretion

King and Dolan[27] have shown that silica which reaches the blood either by absorption from the intestine or the lungs is rapidly excreted by the kidney. The amount appearing in the urine varies with diet and also of course with exposure.

Bloomfield and Goldman[28] followed up the work of King and Dolan by studies on the urinary excretion of silica in a group of 123 anthracite coal miners and found that it averaged 2.5 milligrams per 100 cc of urine, while in a group of 11 control subjects it averaged 1.0 milligrams. They also found that silica is still excreted in the urine of individuals who have been away from any silica dust exposure for several years.

Goldwater[29] studied the urinary excretion of silica in non-silicotic humans and found (1) that there are wide variations in silica concentration depending on the specific gravity of the urine, (2) that the same individual on a constant diet may show wide

daily fluctuations in urinary silica and (3) that different individuals on similar diets show great differences in output. He warns therefore that great caution should be observed in interpreting urinary silica findings.

Silica in Blood and Sputum

Silica may likewise be demonstrated in the blood and sputum. Boehme and Kraut quoted by Sweany,[30] reported a two- to threefold increase in the blood of patients with silicosis. Oranguren and Bartholin[30a] believe that more than 8 mgm. per cent of silica in the blood, as determined by the colorimetric method is significant.

Positive chemical reactions for silica in the sputum may be obtained in all cases of silicosis, but are indicative of exposure rather than diagnostic of the disease.

Reaction of Peritoneal Tissue to Injection of Silica and Other Dusts

It is an interesting fact that a *nodular* reaction of the tissues to silica which in the lungs (shortly to be described) is characteristic of the disease, is not at all limited to pulmonary tissue. Miller and Sayers[31] injected a suspension of various dusts into the abdominal cavities of guinea pigs and found there, after 90 days, reactions similar to those occurring in the lungs upon inhalation of the same dusts. So characteristic were the reactions that this method is recommended by the authors as a practical means of determining the possible harmfulness of any given dust.

The response of the peritoneal tissue to the injection of dusts was of three kinds, namely, proliferation, absorption and inertness. A reaction of proliferation with the formation of nodules was shown to quartz and chert. A reaction of absorption, where the dust deposits tended to disappear was shown to calcite, limestone, calcium carbonate, gypsum and cement—all dusts, it may be noted, containing calcium. Finally a reaction of inertness, where there was neither proliferation nor absorption was shown to soapstone, silicon carbide, ferric oxide, anthracite and bituminous coal and precipitator ash.

McCord[32] and associates confirmed these experiments and determined the peritoneal tissue reaction to a number of other dusts as well.

Reaction of Lymph Nodes to Silica and Other Dusts

The reaction of lymph nodes to suspensions of various dusts was studied by Stüber[33] who found characteristic reactions to dust with a free silica content above 26 per cent. Other dusts such as cornstarch, rouge, manganese, cadmium and silicate dusts with a free silica content of not more than three per cent did not produce these changes. The author suggests that the lymph node injection method may be developed into a rapid means of determining the toxicity of dusts.

Behavior of Silica in the Lungs

The Dust Disposal Mechanism of the Lungs

The body is equipped to deal with inhaled dust in several ways. A good deal of it, especially the larger particles, is caught on the moist mucous membranes of the upper respiratory passages, in the nose, throat, pharynx and trachea and either blown out, coughed out or swallowed. The nose is more efficient in catching dust than the mouth and for this reason mouth breathers suffer more severely than others from the effects of dust.

The finer particles of dust escape retention on mucous surfaces and reach the air spaces of the lungs. There, a more elaborate dust disposal mechanism is brought into play. This depends on two elements, a special type of migrating cell known as a macrophage or phagocyte, and the pulmonary lymphatic system.

The Lymphatic Filter

The latter is composed of a fine network of thin walled lymphatic vessels which accompany all the pulmonary arteries and veins. Through these lymph channels passes a constant stream of lymph from the periphery to the root or hilum of the lungs where it flows through large collections of lymphoid tissue, the tracheobronchial lymph nodes. Small collections of lymphoid tissue, are located at intervals throughout the lymphatic network particularly at the dividing points of bronchi, arteries and veins. Deposits of lymphatic tissue may be thought of as playing the part of filters in the drainage system created by the lung lymphatics. Normally the amount of lymphoid tissue in the lung is least in infancy and tends to increase with age as it is needed. The pleural membrance covering the lungs has its own system of lymphatic channels which communicates freely with that of the lungs proper especially in the septa between lobes.

"Dust" Cells

When dust particles reach the alveoli, the air spaces of the lungs, they are promptly engulfed by the migrating cells already mentioned. Some dusts are engulfed quickly, others slowly. Silica is one of the latter. Some authorities believe that these cells originate from the lining of the air spaces themselves; others from the lining of adjacent blood and lymph vessels. When the "dust cell," as it may be called, has taken up its load of dust it makes its way into the nearest lymphatic vessel and travels along in the lymph stream to a deposit of lymphoid tissue. When the dust cells die the dust particles themselves may be absorbed or they may remain as an inert deposit in the lymph node or they may be picked up by other cells and carried farther. Sometines when lymphoid deposits lie adjacent to a bronchus, dust cells may pass through its wall and a small

amount of dust may work its way out in this way through the bronchi. Unless the lungs are overwhelmed with dust, however, most of the particles reach an ultimate destination in the hilum nodes.

Relatively large amounts of non-irritating dust can thus be taken care of in the lungs without involving much difficulty other than enlargement of the lymph nodes to accommodate it. But with silica dust certain special effects occur.

The Pathology of Silicosis

When silica dust enters the lung its first effect is on the bronchioles, the smallest bronchial passages. It damages their lining cells and causes a slight inflammation which the South African authorities speak of a dry "bronchiolitis." This condition may be at least partly responsible for the dry cough, so common in silicosis.

Silica particles, after reaching the alveoli, are taken up by dust cells in the usual way and carried into the lymph vessels but now, as the engulfed silica becomes dissolved, its particular injurious action begins to take place. The dust cell containing silica may die, or the dissolved or colloidalized silica may penetrate through it. In either case the latter is brought directly in contact with adjacent tissues, and wherever it comes in contact with connective tissue, it stimulates increased growth and causes a condition of fibrosis or overproduction of connective tissue to develop.

Growth of Connective Tissue

This reaction is conspicuous first in the tracheo-bronchial lymph nodes. The sinuses or spaces through which the lymph flows in the nodes become gradually obstructed by the growth of connective tissue. The flow of lymph is slowed down until an actual backing up may occur. Then the dust cells with their silica loads settle in all the deposits of lymphoid tissue and, wherever they collect an overgrowth of connective tissue follows. As the resulting obstruction increases, the dust cells which Gardner has found are rendered more than usually active by the presence of silica, tend to penetrate through the walls of the lymphatic channels into the lung tissue and here again connective tissue overgrowth is set up.

The result is a generalized fibrosis which affects the alveolar walls, interferes with the elasticity of the lungs and encroaches on air spaces and small blood vessels. The same process is set up in the pleura overlying the lungs when backing up of lymph carries dust cells and silica into the pleural lymphatics. The result here is a dry pleurisy with thickening and adhesions.

The Silicotic Nodule

The form which connective tissue overgrowth produced by silica takes is characteristic. Its feature is the development of a definite

nodule sometimes called a pseudotubercle, which is somewhat similar to the tubercle of tuberculosis but with definite differences. Gardner[11] has described in detail the formation of these nodules in guinea pigs where the lesion is essentially the same as that found in man.

Figure 1

Microphotograph of Section of Lung Tissue Showing Silicotic Nodule

(From "Silicosis", International Labor Office, 1930)

The nodules begin in collections of lymphoid tissue where the phagocytes or dust cells are arrested and the shape of the nodule, whether round, oval or elongated, depends on the configuration

of the lymphoid deposit. The irritating effect of the silica stimulates the growth of connective tissue cells which gradually replace the lymphoid cells. These connective tissue cells became arranged in a whorl-like manner at the center, while at the circumference they take on a laminated or layer-like arrangement. Gradually the cellular character at the centre is lost and in the fully developed nodule, the centre is composed of clear hyaline material with a surrounding zone of connective tissue. Caseation or calcification of the centre of the nodule may occur.

Growth of the nodule occurs at its circumference and proceeds as the disease progresses, nodules becoming both larger and more numerous. Individual nodules may reach the size of 2–5 mm. or slightly larger, and be easily palpable and visible to the naked eye. Composite nodules may be formed by the growth of adjacent, individual ones. Contrary to the behavior of tubercles, breaking down the simple silicotic nodules does not occur. When it does it is an indication that infection has been added to the silicotic process.

When nodules form under the pleura they give the appearance of small, whitish, raised areas spoken of as "sub-pleural plaques." Not all the fibrosis in silicotic lungs is nodular. A fine diffuse fibrosis occurs also, and the extent to which the nodular or the diffuse type predominates seems to vary in different occupations depending possibly upon the percentage of free silica in the dust breathed, the rate of development of the disease, or other unknown factors.

Tuberculosis with Silicosis

The same mechanism by which dust is disposed of in the lungs operates to take care of infectious organisms. Tubercle bacilli and silica particles are engulfed by phagocytes in the same way. Consequently the silicotic and tuberculous processes are closely associated, and the lesions of the two show some similarity in their distribution.

The presence of the two combined conditions modifies the characteristic appearance of each. Tuberculous lesions in a silicotic lung show more fibrosis than would ordinarily be expected and silicotic nodules in the presence of a tuberculous infection tend to break down and become caseous as they do not do in simple silicosis. The large areas of massive consolidation found in advanced silicosis are often the result of the combined effect of silica and the tubercle bacillus. Such areas are, as a rule, larger than are formed by the coalescence of adjacent nodules in uncomplicated silicosis. Tubercle bacilli may be found in them, but they are notoriously hard to detect. The nodules may show caseation and cavitation, though excavation is less frequent than in uncomplicated tuberculosis.

Some authorities believe that a low-grade, latent tuberculous infection may be present in certain silicotic lesions from the start when tubercle bacilli are inhaled along with the dust, but that it

remains enclosed in fibrosis and quiescent until activity develops later. Such a condition usually occurs in the apical region while the rest of the lung may be either free from obvious silicosis or show it in an early stage. In other cases larger irregular nodules scattered here and there throughout a silicotic lung may be produced by a latent tuberculous infection without any clinical signs. Animal inoculations from such lesions have, according to Strachan and Simson,[34] been positive in a considerable number of cases.

Active tuberculous infection may occur at any stage of silicosis but is more common in the later stages when it usually runs a chronic course. The acute types, tuberculous pneumonia and miliary tuberculosis, are uncommon in the later stages of silicosis probably because of the more extensive fibrosis. In the early stages of silicosis, tuberculosis ordinarily runs its usual course but appears definitely to aggravate the silicosis. Sometimes the onset of a tuberculous infection, according to the authorities quoted above. brings to light signs of silicosis which were previously not detectable. In such cases the infection seems to select sites in the lung where minute aggregations of silica are already present and to assume the characteristic miliary distribution of silicosis. Willis[35] states that as a rule tuberculosis infection with silicosis may occur in two forms, i.e., (a) apical and unilateral with downward spread as in ordinary pulmonary tuberculosis, and (b) non-apical, often bilateral, asymmetrical and often massive. Gardner[1] suggests that the apical lesion represents an infection, present before exposure to dust, which has failed to heal under its influence; while the non-apical form may represent a new, truly industrial infection. While massive areas of fibrosis usually occur late, in a few cases they may be present from the first, especially in the apical regions.

Evidently two factors operate to influence the development of tuberculosis in the silicotic lung. One is the coincident fibrosis which tends to limit and enclose the infection. The other is the tissue injury produced by silica which tends to reduce resistance to infection. Moreover the fibrosis, while it may limit the spread, may also tend to prevent healing and possibly result in more numerous and larger infected areas.

Belt[21] suggests that tuberculous infection gets its start primarily where collections of silica particles occur in regions of less adequate blood supply. There the tissue becomes devitalized by the action of silica while the blood supply is inadequate for defense or repair. Relatively avascular areas, according to him, become more and more numerous as fibrosis progresses and it is this which accounts for the greater incidence of tuberculosis in advanced than in early silicosis.

Post Mortem Appearance of Silicotic Lungs

The first thing to look for in silicotic lungs according to authorities already quoted, is evidence of fibrosis first in the root glands, second in the pleura and third in the lung substance. Root glands

are enlarged, firmer than normal and pigmented according to pigment in the dust breathed. The visceral pleura shows pigmentation and thickening together with a variable number of nodules or plaques which can be felt beneath the surface and appear as fine pin point pearly white areas surrounded by pigment.

On section, the surfaces of the lungs show small pigmented nodules. These can not be felt in the earliest stages. Later they become palpable and project above the cut surface. Their number and size give an indication of the stage of the disease. Thus, according to Strachan and Simson,[34] in early silicosis they may be small and moderately numerous or medium sized and sparse; in moderately advanced silicosis they may be small and numerous or large and moderately numerous; while in advanced silicosis they are both numerous and large. Nodules up to two mm. in size are considered small, from two to four mm. medium, and from five mm. to a centimetre, large.

Willis[35] summarizes the appearance of the lungs in cases of well developed silicosis as follows:

> "The gross appearance of the silicotic lung is characteristic. It is stiff, inelastic, pigmented and adherent. It offers resistance to the knife which cuts it with a grating noise, the incision yielding a mottled surface of pigmented nodule, often grown around a bronchus or blood vessel, much pigment, diffuse fibrosis, thickened interlobar septum and thickened bronchial and vascular walls. Occasionally in the absence of complicating tuberculosis and often in the presence of the latter, there may be large densely fibrotic masses which occupy much of a lobe or a lung and which may be caseous or cavitated at the centre."

The lungs may not be much increased in size but show an increase in weight. Emphysema, particularly in the marginal regions and at the apices, is a more or less constant feature. In some cases plaques occur on the diaphragmatic pleura. Occasionally silicotic fibrosis may be found in neighboring lymph nodes in the posterior mediastinum, at the cardiac end of the stomach, around the pancreas and at the hilum of the liver.

In advanced stages, bronchitis is well marked and the bronchi are obviously thickened as are also the interlobar septa and trabeculae. Alveolar fibrosis is not marked except when infection is present or dust exposure has been overwhelming.

Tuberculous infection superimposed on silicosis is very difficult to detect by appearance in the early stages. Nodules may be more numerous at the usual sites of election for tuberculosis. In well marked cases of tuberculo-silicosis the root glands are much enlarged, of a grayish color, sometimes with foci of caseation, or calcification. There are dense adhesions on the pleura with much scarring and patchy thickening. Caseation can be seen in the nodules which in certain areas, especially under the pleura and

toward the upper parts of the lobes, may become confluent with breaking down of tissue and cavity formation.

Examination of Lung Tissue for Silica

The actual presence of silica in the lungs can be determined by chemical analysis after ashing. The results are expressed as the percentage of SiO_2 in the total ash of the lung, or as milligrams per gram of dried lung tissue.

According to McNally,[36] more than two milligrams of SiO_2 per gram of dried lung tissue is evidence of undue exposure. The normal level being about one milligram. With two or more milligrams, nodulation is usually present but the degree is not proportional to the amount of silica present.

Silica is present in higher concentration in the lymph nodes. The normal for city-bred adults is approximately six milligrams per gram of dried tissue.

Crystalline silica fragments can also be seen in lung tissue by examination under the microscope with polarized light. But there is no relation between number of particles seen in a given area and the chemical content of SiO_2 since the silica ceases to be visible when it loses its crystalline form.

Symptoms and Course of Silicosis

Rate of Development

Silicosis develops slowly under ordinary conditions. Pancoast and Pendergrass[37] found that the earliest cases of advanced silicosis among granite cutters occurred after 10 to 14 years; among pottery workers, after 24 years; and among metal grinders after five years. On the other hand they found cases of early silicosis in granite cutters after 20 years; pottery workers after 35 years, and metal grinders after 37 years. Under exceptionally severe exposure it develops in less than a year's time. The development of the disease depends on the extent of the exposure and the individual's reaction to dust. Seven years is probably about the usual time it takes to develop silicosis under conditions of moderate severity.

Predisposing Factors

Susceptibility varies with conditions such as nasal obstruction, infection and, possibly, age. When a man breathes through his mouth he loses the advantage of the extensive dust catching mucous surfaces of the turbinate bones and inhales more dust than he otherwise would. South African investigators have found that mouth breathers are definitely more subject to silicosis than nose breathers. Lehmann[38] says "the susceptibility of workers with poor nasal filtration is much greater than that of men with good nasal filters."

Chronic lung infection is likely to predispose to silicosis through involvement of the lymphatic system, thus facilitating lymphatic

obstruction. A tuberculosis infection especially renders an individual more susceptible to the effects of siliceous dust, and its onset may bring to light, in a comparatively short time, evidences of previously unsuspected silicosis. It is possible that silicosis develops more slowly in a young person than in an older one because of the less abundant lymphoid tissue present in the lungs in youth to catch and hold dust, but there is as yet no clear evidence on this point.

Symptoms

An individual who is developing silicosis may not know, even when extensive changes have taken place in his lungs, that there is anything wrong with him. Approximately one-fourth of the men in a group of New York City rock drillers whose chest X-rays showed advanced silicosis had no complaints whatever. When symptoms do occur, shortness of breath is likely to be the first one noticed. This becomes progressively more marked as the disease advances, until in the late stages it may be completely incapacitating. With the dyspnoea there is likely to be some cough, usually dry, unless infection is present and some pain in the chest from pleural irritation. There may also be some epigastric pain. There is usually no loss of weight in simple silicosis and no fever or malaise.

The frequent association of tuberculosis with silicosis has already been mentioned and it was stated that while it may occur at any stage of development of the disease it is more common in the late ones. Individuals are found whose X-ray pictures show clear evidence of tuberculosis with silicosis who yet remain without symptoms; but eventually, when the infection becomes clinically active, evidences of toxemia such as fever, loss of weight, fatigue and night sweats make their appearance.

Course

The course of silicosis differs greatly in different individuals and under different conditions of exposure. It may develop so slowly that at the end of 40 or 50 years of dusty industrial life an individual may show only the earliest stage. It may develop in as short a time as one or two years under very severe conditions. It may apparently remain quite stationary even with continued exposure of a mild degree. On the other hand it may continue to progress even after exposure has ceased if the total dust dose has been severe enough. When tuberculosis is superimposed or becomes active in an advanced stage of silicosis, progress is usually rapidly down hill. This is borne out by the U. S. Public Health Service experience with the granite cutters at Barre, Vermont, where it was found that the median duration of cases of silico-tuberculosis, from the time of disabling sickness to death, was 15 months.

Physical Signs

Just as there may be few or no revealing symptoms until a late stage has been reached so the physical signs may be indeterminate and unreliable.

The appearance of an individual with silicosis is usually good. These people tend toward a robust type and are as a rule overweight. The first significant sign may be a diminution in the chest expansion which becomes progressively less as the disease develops and may be reduced to as little as two centimeters from the normal nine. The chest tends to be rigid and barrel shaped as in emphysema. In advanced cases there is usually some cyanosis and clubbing of the fingers.

In addition to restriction in chest expansion other evidences of functional impairment are to be found in an increased respiratory rate immediately after exercise; an increased lapse of time before the pulse returns to normal after exercise and diminution in the vital capacity.

Examination of the lungs reveals diminished resonance and distant breathing. The frequent presence of emphysema, however, often results in increased resonance. British investigators especially describe a harshened, thinned and shortened inspiratory murmur as characteristic. Râles are not heard as a rule unless infection is present. The chest is spoken of as characteristically "dry."

With Superimposed Tuberculosis

The first sign of active tuberculosis may be a definite and progressive loss of weight. The patient's "temperature rises and his pulse rate accelerates, his cough becomes definitely increased and his sputum more abundant and often streaked with blood; pleurisy is common; asthenia and exhaustion become extreme and the patient dies."[35] Gardner[9] finds that the sputum in silicotuberculosis is usually negative for tubercle bacilli until late in the disease.

There are no characteristic changes in the blood picture, blood pressure or urine, except possibly increased excretion of silica. The red cell count and the hemoglobin may show a compensatory increase.

X-ray Appearances

Early

In order to understand the X-ray appearances of silicosis, it is important to remember the underlying condition, the increase of fibrous tissue in the lung. It is this which is responsible for all that the X-ray shows.

At the very beginning of the process there is an increase in the normal lung markings similar to that found in chronic bronchitis

or chronic passive congestion of the lungs. The hilum shadows are increased in extent and prominence; there is an increase in the trunk shadows and an increased prominence of the linear markings in the peripheral zone. While these changes will be found to develop under observation in a normal lung upon exposure to silica, they are not characteristic of that condition, occurring as they do from other causes and hence they have no general diagnostic value. The British speak of this appearance as "large branch fibrosis," likening the lung markings to the outline of a tree. As the disease progresses the fibrosis becomes more generalized. It is apparent beyond the main branches of the bronchial tree, and suggests the appearance of fine branches and twigs. This has been called small branch fibrosis, or "arborization." It is still not definitely diagnostic of silicosis.

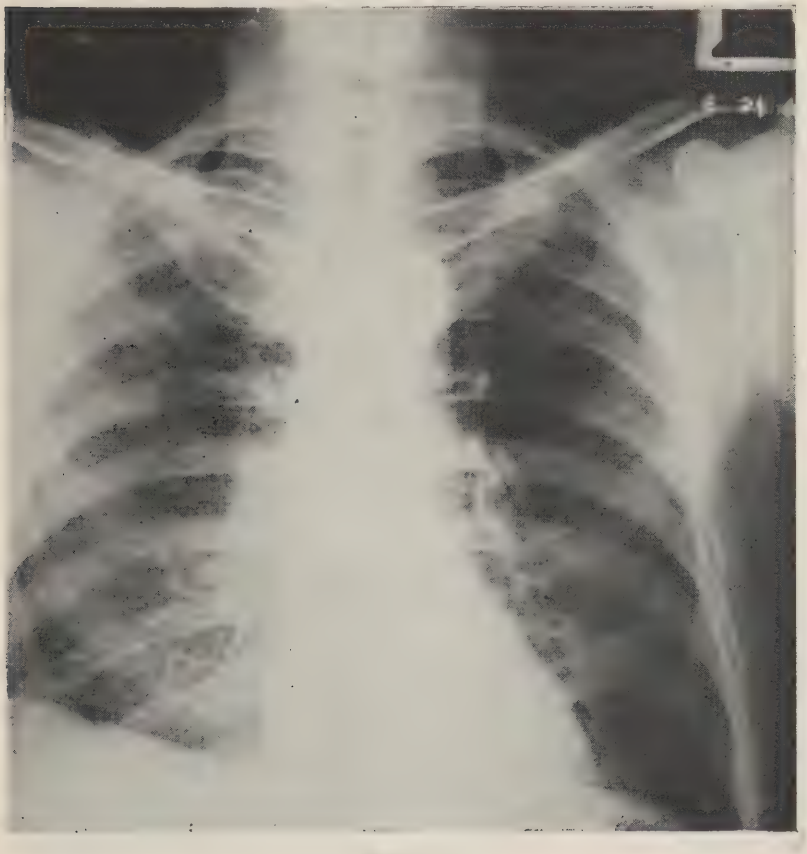

Figure 2

Silicosis Stage One—Calcimine Worker—Exposure 30 Years

Nodulation

It is not until the next development, the appearance of definite mottling due to the formation of silicotic nodules large enough to cause definite shadows that the picture can be considered characteristic. This appearance is the first specific sign of silicosis. It has been likened to a "leafless tree putting on leaves."

The mottling in silicosis usually appears first on the right side about the root of the lung. Later it becomes generally distributed throughout both lungs, though still with a tendency to be more marked at the roots and less in the apical regions and bases. The nodules vary in size from two to six millimeters and increase in size and number as the disease progresses. In the most advanced form of silicosis, nodules coalesce forming large, irregular masses. Massive, conglomerate lesions probably develop, according to Gardner,[9] in areas where the lung tissues have been injured by previous infections that have subsequently healed.

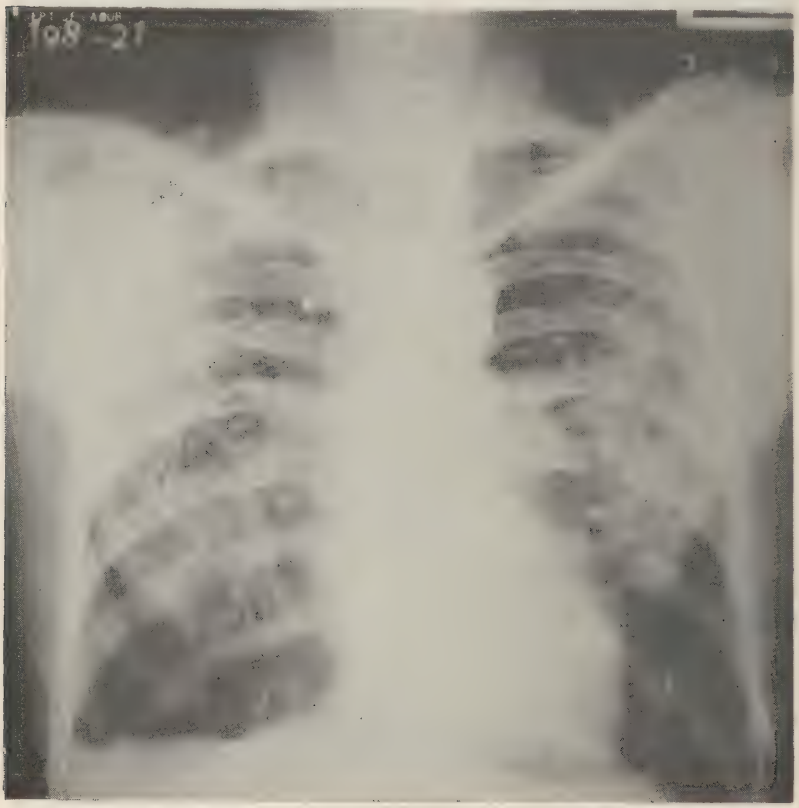

Figure 3

Silicosis Stage Three—Miner—Exposure 14 Years

Interstitial Fibrosis

This is the typical, though not the invariable, appearance of silicosis. A type with much interstitial fibrosis as well as nodulation may occur and is apparently commoner in some types of industrial exposure than others. Pancoast[12] relates variations in X-ray appearances to the underlying pathology, pointing out that the linear fibrosis follows blockage of the lymph channels; the nodular type depends on fibrosis occurring in lymphoid deposits, and the interstitial type on penetration of interstitial tissue by dust cells, presumably occurring when the lymphatic block is rapid.

Silico-Tuberculosis

Gardner[13] describes the superimposition of tuberculosis upon silicosis as follows:

> "It may manifest itself as simple superimposition or as a new type of disease known as silico-tuberculosis. The primary

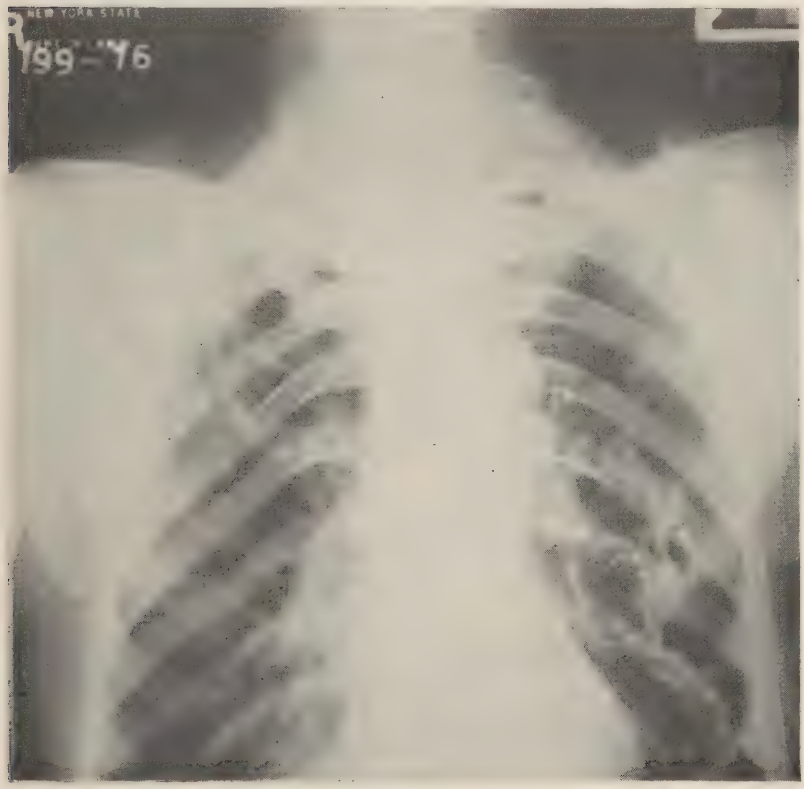

Figure 4

Pulmonary Fibrosis with Tuberculosis and Pleural Calcification in a Tremolite Talc Worker—Exposure 16 Years

complex of tuberculosis can be detected on a background of generalized nodulation. The silica rarely has any effect upon these foci as they are generally sterile by the time persons begin to work in dust. Very rarely they have been reactivated. The scars of healed apical tuberculosis are recognizable in the silicotic lung. After prolonged exposures to dust such lesions have been seen to break down and give rise to very chronic silico-tuberculosis, a conglomerate type of disease, that spreads slowly from the vicinity of the original focus harboring bacilli. This lesion is productive rather than exudative in character; cavity formation, if it occurs, is a late manifestation. Aspiration and bronchogenic spread to remote portions of the lungs are limited. Silico-tuberculosis is much more commonly found in the base of the lung than simple tuberculosis. Whether the basal lesion of the silicotic is a manifestation of exogenous reinfection or whether it originates from reactivated latent foci has not been determined.

"The focus of silico-tuberculosis may be difficult to differentiate from the conglomerate focus of simple silicosis, but the former being due to an active infection is constantly, though slowly, changing. Repeated examinations by all available methods are essential for a diagnosis.

"Miliary tuberculosis occurs as a terminal event but it is particularly difficult to detect in the roentgenogram showing generalized discrete nodulation."

Standard Terminology Suggested by U. S. Public Health Service

There has in the past been considerable diversity in the interpretation of films showing the effect of dust exposure. This is of course inadvisable and for the sake of promoting greater uniformity the U. S. Public Health Service[44] has set up a tabulation of X-ray appearances with the underlying histologic changes. It is hoped that this will be accepted as the recognized standard for interpretation. The tabulation is reproduced below:

Roentgenological Appearances	Histological Appearances
HEALTHY LUNGS AND ADNEXA	
1. Healthy lungs. As defined by the NTA Committee report.	1. Essentially the normal tissues of the vascular tree, the mediastinum, the bronchi, and trachea.
2. Irregular exaggeration of the linear markings, with possibly some beading confined to the trunks.	2. Cellular connective tissue proliferation about lymphatic trunks in the walls of vessels and bronchi. Beading may be due to various causes, as blood vessels seen end on, arteriosclerosis, minute areas of fibrosis in lymphoid tissues along the trunks.
3. Increased root shadow.	3. Cellular reaction in the tracheo-bronchial lymph nodes with extensions along afferent lymphatic trunks.

These changes come within normal variations when not accompanied by recognized organic disease.

SIMPLE SILICOSIS

4. Nodulation. Discrete shadows not exceeding six mm. in diameter, tending to uniformity in size, density, and bilateral distribution, with well-defined borders surrounded by apparently normal lung shadow. The outer and lower lung fields characteristically show fewer nodules.	4. Circumscribed nodules of hyaline fibrosis located in the parenchyma of the lung. Occasionally some of these nodules may show microscopic foci of central necrosis.
5. Conglomerate shadows that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by: a. Localized increased transparency of the lung with loss of fine detail. b. Intensification of the trunk shadow by contrast. c. Depression of the domes with possible tendency toward individualization of the costal components of the diaphragm. d. Lateral view: Increase in the preaortic and retrocardiac space with exaggerated backward bowing of the spine. Widening of the spaces between the ribs may or may not be present.	5. The result of coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more or less hyaline fibrous tissue. The lung architecture is partially obscured. No demonstrable evidence of infection. Emphysema is a compensatory dilation of the air spaces with or without thickening of the septa.

SILICOSIS WITH INFECTION

The characteristic appearances described under simple silicosis are modified by infection as follows:

6. Localized discrete densities and/or string-like shadows accompanying those of simple silicosis described above.	6. Strands of fibrous tissue, often along trunks and septa, with or without areas of calcification; indicative of "healed" infection.
7. Mottling. Shadows varying in size with ill-defined borders and lacking uniformity in density and distribution, accompanying simple silicosis.	7. (a) Areas of broncho-pneumonia with or without caseation, i.e., acute infection. (b) Lobular areas of proliferative reaction with or without caseation, i.e., chronic infection.
8. Soft nodulation. The nodular shadows described under simple silicosis, 4, have now assumed fuzzy borders and/or irregularities in distribution. This change may or may not accompany the simple mottling of 7.	8. Perinodular cellular reaction either exudative or proliferative in character.
9. Massive shadows of homogeneous density not of pleural origin symmetrically or asymmetrically distributed.	9. Extensive areas of fibrosis probably due to organized pneumonia of tuberculous or nontuberculous origin superimposed upon a coexistent silicotic process. Outlines of normal structures may be partially destroyed.

Complications of Silicosis

Pulmonary

In addition to tuberculosis and pleurisy, which are the commonest and the most important complications of silicosis, certain other associated conditions are of interest. A tendency to recurrent chest colds with prolonged cough is rather characteristic. Some authorities believe that chronic bronchitis is present in all cases where the disease is well advanced. Collis and Yule[8] in their statistical study found that the death rates for all non-tuberculous respiratory infections were higher among workers in siliceous dusts than in the general population.

Bronchiectasis, lung abscess and gangrene occur frequently in hard-rock miners, according to Proske and Sayers[38a] who believe that prolonged inhalation of silica dust, contaminated with organisms from the mouth, paves the way for bacterial attack upon the bronchial mucous membranes.

Emphysema is a common complication of silicosis and tuberculosilicosis and is often of the bullous type along the lung margins and in the apices.

Cardiac

Tuberculous pericarditis may sometimes arise in silico-tuberculosis by a backward lymphatic spread.

The question of the extent to which cor pulmonale (right-sided enlargement of the heart) occurs in silicosis is debated. Coggin, Griggs and Stetson[38b] report finding exclusive right ventricular hypertrophy in 44.1 per cent of 102 autopsies on cases of pneumoconiosis, and hypertrophy of both ventricles in an additional 14.7 per cent. Electrocardiographic studies in 43 cases showed right axis deviation in 37.2 per cent.

Gardner[38c] questions whether simple nodular silicosis is associated with hypertrophy of the right heart and believes that the cardiac symptoms which are often late manifestations of massive conglomerate (third stage) silicosis result from arteriosclerosis and emphysema as well as the fibrosis.

Silicosis and Cancer

The question of whether inhalation of silica dust or the presence of silicosis predisposes to lung cancer is brought up from time to time but there is no conclusive evidence that this is the case. Occasionally the co-presence of the two conditions is striking. Dible,[39] for instance, reported three cases of malignant disease in 14 post mortem examinations of silicotic subjects.

On the other hand, Berblinger[40] reports that in 82 autopsied cases of lung cancer he has never found silicosis, and Teleky[41] points out that were there any connection between the two conditions it would certainly have been brought out in the extensive South African experience. There, however, six cases of lung cancer were found in 1,109 autopsied white miners without silicosis, six cases in 1,023 autopsied Europeans, not working in mines and four cases in 1,083 autopsied white silicotic miners. This experience would seem to establish definitely the absence of causal relationship.

Diagnosis

In making a diagnosis of silicosis, a history of the individual's dust exposure and symptoms is of primary importance, and next to this a well taken X-ray of the chest. The physical examination,

many think, is least important because of the variability and frequent absence of signs. However, it is useful in determining the presence of infection.

X-ray Technique

For the X-ray examination a rapid exposure is desirable. With 100 milli-amperes an exposure of 1/10 of a second at a 66-inch target distance, varying the kilo-voltage according to chest thickness, gives very good results.

Early Silicosis

Some difficulty may be experienced in the diagnosis of early silicosis. As already stated, the increase in linear markings occurring at the beginning of a silicotic process bears no distinguishing features, and silicosis cannot be diagnosed until some true nodulation appears. Nodulation in its early stages may sometimes be simulated by fibrosis from some infectious process. In such cases the history of dust exposure, of past respiratory disease and of complaints may be decisive.

Advanced Silicosis

In its more advanced stages silicosis may very closely resemble miliary tuberculosis, but here again the story of the silicotic in relatively good health with few symptoms will contrast strikingly with that of the patient suffering from miliary tuberculosis. Fungus infections of the lungs may at times give an appearance indistinguishable from silicosis as may sarcoidosis. In such cases examination of sputum by Irwin's[45] microincineration technique would demonstrate the presence or absence of silica. In medico-legal cases which have been fatal, determination of the amount of silica in lung tissue is of value.

Silico-Tuberculosis

The diagnosis of tuberculosis superimposed upon early silicosis does not usually present much difficulty because the X-ray appearances of tuberculosis are not yet obscured but the question of whether in a case of obvious tuberculosis early silicosis is present may be a more difficult one, impossible to determine except upon autopsy. The presence of tuberculosis when silicosis is in the well developed nodular stage may be suspected by the appearances already discussed. Willis[35] says "it is very helpful to remember in this connection that the pneumoconiosis rarely affects the extreme apices as tuberculosis does."

In the most advanced stage of silicosis with diffused fibrosis or massive consolidations it is very difficult in many cases to know whether tuberculosis is also present. Serial studies should then

be made to show progress and a careful search for evidences of cavitation and for tubercle bacilli in the sputum. Ischaemic cavitation may, however occur in simple silicosis.

Classification According to American Public Health Association

For the sake of clarity in diagnosis and also for the purpose of aiding in compensation settlements, various classifications of the stages of silicosis have been made from time to time. Some of these have been based primarily on X-ray findings; others on a combination of X-ray and clinical findings, and others on a roentgenopathological basis. They differ from each other considerably and enumeration of them all could only be confusing. The classification suggested by the Committee on Pneumoconiosis of the American Public Health Association[46] is given below:

"**First stage** (corresponds to anteprimary stage of South Africa):—The symptoms of uncomplicated first-stage silicosis are few and often indefinite. The man may apparently be quite well and his working capacity not noticeably impaired. Slight shortness of breath on exertion and some unproductive cough, often with recurrent colds, are the most usual symptoms. The man may have a little less ability to expand his chest than formerly, and the elasticity of the chest may be slightly impaired. The earliest specific indication of the presence of silicosis is the radiographic appearance, consisting of generalized arborization throughout both lung fields with more or less small, discrete mottling.

"This characteristic mottling is due to shadows cast by the discrete individual nodules of fibrous tissue in the lungs, and is essential to the diagnosis of silicosis; without this finding the diagnosis of silicosis is not sustained except by autopsy.

"**Second stage** (corresponds to primary stage of South Africa):—A definite shortness of breath on exertion is usually found, and pains in the chest are a frequent complaint. A dry morning cough is often present, sometimes with vomiting, and recurrent colds are more frequent. Even then the man's appearance may be healthy but he is dyspnoeic on exertion; he can not work as well as formerly; his chest expansion is noticeably decreased, the movement being sluggish and diminished in elasticity.

"The characteristic radiographic appearance is a generalized medium-sized mottling through both lung fields. The shadows of the individual nodules are for the most part discrete and well defined on a background of fibrous arborization, but there may be here and there larger but limited opacities due to a localized aggregation of nodules.

"**Third stage** (corresponds to the secondary stage of South Africa):—In the third stage the shortness of breath is marked and distressing even on slightest exertion. The cough is more

frequent; the expectoration is in most cases slight but may be copious. The individual's capacity for work becomes seriously and permanently impaired; his expansion is greatly decreased even with forced inspiration; he may lose flesh; his pulse rate may be increased and his heart may become dilated.

"The radiographic appearances in the third stage are further accentuated; the mottling is more intense; the nodules are larger, of a conglomerate form so that large shadows are shown corresponding to areas of dense fibrosis.

"Physical examination of an individual may reveal changes in percussion and ausculation, mild in the first stage and increasing with the progress of the disease. These alone are not sufficient to be of great value in the diagnosis of silicosis."

Management of Silicosis

There is no cure for silicosis in the sense that the fibrosis may be made to disappear. Simple silicosis is seldom disabling and as a rule individuals suffering from it are better off at their accustomed work though every effort should be made to reduce their exposure to a minimum. When tuberculosis is superimposed the situation changes. Gardner[1] makes the following suggestions:

"No person with open tuberculosis should be permitted to work in an industry where silica dust is created. An old employee with closed silico-tuberculosis that does not incapacitate him may be allowed to do light work in departments where no dust is generated. Sanatorium treatment may be tried but it is often ineffective. A young employee with silico-tuberculosis should be given the benefit of sanatorium treatment.

"Persons over 40 with roentgenographic evidence of well-healed adult type tuberculosis can be employed in an industry with a silica hazard. Their only danger lies in the development of a massive conglomerate type of fibrosis. But it is assumed that an industry that is having pre-employment examinations is cognizant of the hazard and is making every effort to reduce the dust concentrations in its plants.

"The younger the individual with X-ray evidence of healed adult type tuberculosis, the less safely can he be exposed to silica dust.

"Roentgenographic evidence of a healed primary complex in a person over 16 years of age does not constitute grounds for disbarring him from exposure to silica."

Use of Metallic Aluminum

Interesting results in the treatment of silicotics with freshly ground metallic aluminum have been recently reported by Crombie, Blaisdell and MacPherson[46c] (see page 64 for further discussion).

Daily inhalation of aluminum powder by a group of 34 men with simple silicosis and measurable pulmonary disability was found to result in a diminution of dyspnoea, cough, pain and fatigue in 55 per cent.

Acute Silicosis

Under conditions of very intensive exposure to dust of an extremely high silica content, death has been reported in as short a time as one to 17 months after the beginning of exposure. Such cases have been referred to as "acute silicosis."

Kessler[47] reported six such fatal cases occurring in a plant where sand was pulverized to fine powder for use as an abrasive. The free silica content of the sand was approximately 99.24 per cent. All of the cases developed after exposure of from four to 18 months.

Chapman,[48] in 1932, reported three cases of silicosis, two of them fatal, occurring in men who worked at machines where powdered silica and alkali were mixed for the making of abrasive soaps. In one case symptoms developed after eight months of exposure, in another after 29 months. The third was incapacitated for work in a little over two years.

Kilgore[49] in the same year reported four cases of silicosis, three of them fatal, in men who worked in a polishing powder plant where quartz was pulverized on the premises. None had had an exposure over 14 months. Death had occurred 50, 21 and 14 months respectively after the beginning of exposure.

At Gauley Bridge, West Virginia, in 1929 a tunnel was constructed through rock with a silica content of 97 to 99 per cent. Under such conditions the dust hazard was severe. Many men* out of approximately 2,000 exposed, died in a period of some five years following the commencement of the work; death in these cases being attributed by some authorities to so-called "acute silicosis," while others attributed it to tuberculosis, or other respiratory disease.

Gardner[51] examined at autopsy 15 such cases and found that infection had been the cause of death in all. Tuberculosis was definitely the cause of death in 11, probably the cause in two and unresolved pneumonia was the cause in two. Microscopic, but not macroscopic lesions of silicosis were present. Three characteristic changes were seen, namely: (1) Masses of small nodules in broad sheaths of fibrous tissue surrounding the pulmonary lymphatics; (2) Generalized fibrosis of the alveolar walls, an appearance usually seen only in advanced cases of silicosis; and (3) no, or slight involvement of the mediastinal lymph nodes.

The X-ray appearances of these cases were described by Sampson.[52] He mentions two types, the first showing tuberculosis only, and the second tuberculosis with diffuse, fluffy nodulation.

---

* Exact figures are not obtainable. See Congressional Record 50 H. J. Res. 449, 74th Cong., 2nd Session.

. Acute silicosis appears rather to be either acute infection or the acute exacerbation of previously existing infection, resulting from the effect of overwhelming doses of dust of a high quartz content.

Asbestosis [53]

Symptoms

The disease produced by the inhalation of the silicate asbestos is characterized chiefly, but not invariably, by cough, dyspnoea, scanty expectoration, slight cyanosis and emaciation. It develops slowly, as a rule, over a period of 10 to 20 years, though fatal cases have occurred in five years time.

Physical Signs

Physical signs when present are those of a basal fibrosis with diminished breath sounds, limited chest expansion and diminished resonance. Fine dry râles and pleural friction sounds may be present. Risk of superimposed tuberculosis is less than with silicosis, though fatal termination from the fibrosis alone is more common than with silicosis.

Pathology

The pathology of the disease is definitely different from that of silicosis. The asbestos fibres are not easily ingested by dust cells and removed to lymphoid tissue, but remain in contact with the walls of the air spaces and set up a fibrosis which begins about the terminal bronchioles forming a sort of "collar" of fibrous tissue and spreads to form diffuse patches in the parenchyma. Infection, superimposed on asbestosis, as with silicosis, causes a more severe reaction.

Evidence that asbestosis is associated with an increased incidence of pulmonary cardinoma has been presented by Teleky[53a] and also by Wedler.[53b]

X-ray Appearance

The X-ray appearance in asbestosis is described as a fine pinpoint mottled "ground glass," "veiled" or "blurred" appearance, commencing at and involving principally, the bases of the lung.

Diagnosis

Diagnosis is made by the history and X-ray findings and is facilitated by finding microscopically in the sputum clumps of so-called asbestos bodies, yellowish, elongated bodies with bulbous ends. These result from the deposition on asbestos fibres of iron in the form of iron silicate.

The incidence of the disease depends upon essentially the same factors of intensity, duration of exposure, etc., as does silicosis. In this country owing to the relatively small numbers employed in work involving exposure to asbestos and to the relatively good plant hygiene it does not present a serious problem.

Talc Pneumoconiosis

A disabling form of diffuse pulmonary fibrosis caused by the inhalation of tremolite talc dust has been reported by Siegal, Smith and Greenburg.[53c] An unusual feature of talc pneumoconiosis is the development of extensive pleural calcifications.

CHAPTER IV

INVESTIGATIONS OF SILICOSIS

Summary of Silicosis Investigations in the United States

It is impossible of course to estimate how many cases of silicosis there are in the United States. By the nature of the disease, most of them are unknown even to the subjects. But more and more information as to the risk of its development in various dusty trades is being assembled by special investigations. Most of these have been conducted by the United States Public Health Service but State Departments of Health and Labor, Universities and other agencies have done their share.

A resume of the more important studies is given below. A word of caution is necessary in comparing the findings. Standards of diagnosis for the earlier stages of the disease have varied considerably in the past among different interpreters. At one time there was a tendency to include cases showing marked increase of linear fibrosis in the early silicosis groups. As the disease has become more familiar, interpretation of early changes has become more conservative.

Asbestos

Lanza, McConnell and Fehnel[54] in 1935 reported on the examination of 126 persons employed in asbestos plants in the United States. Of the total number, four were diagnosed as having second degree asbestosis, 63 as first degree, 39 as doubtful and 20 as negative. Only one case of active tuberculosis (diagnosed by X-ray) was found. Cases of definite cardiac enlargement were frequent. Dust counts ranged from ⅓ to 82 million particles per cubic foot. Particles in size up to 360 microns in greatest diameter were counted.

During the same year, a report on asbestosis in fabricating plants was published by the Department of Labor and Industry of Pennsylvania.[55]

Dust concentrations in various departments ranged from 0.3 million particles per cubic foot of air to 123.3 million. Particle size was found to range from 1.35 to 2.12 microns in longitudinal diameter and from 0.45 to 0.69 microns in transverse diameter. No free silica was found. Fourteen, or 25 per cent, of 56 workers examined had clinical and roentgenological evidence of asbestosis.

In 1936, Donnelly[55a] reported on 151 asbestos workers of whom 52 showed asbestosis and in the same year McPheeters[55b] reported the examination of 210 asbestos mill workers of whom 25 per cent showed questionable or definite asbestosis.

Cement

In 1928 a study of the health of cement workers was reported by the U. S. Public Health Service.[56] This study was conducted in a representative Portland cement plant and included an analysis of the causes and severity of sickness among a group of cement workers; a study of the physical condition of cement workers by means of physical examinations and X-ray films, and a study of the working environment, especially the character and amount of dust to which the workers were exposed.

The cement was composed of 62 per cent lime and 22 per cent silica together with small amounts of aluminum oxide, magnesia and sulphur trioxide. Free silica in the form of quartz was present in amounts varying from one to 6.5 per cent.

Dust counts in dusty locations ranged from 22 to 92 million particles per cubic foot. The dustier occupations of the industry were found to be associated with a high rate of disease of the upper respiratory system, skin, eyes, ears and digestive system. Of 53 individuals who were X-rayed, 15, or 28 per cent, showed evidence of lung damage from dust but of a relatively mild degree, since none of these had any symptoms of the condition. Tuberculosis was present in three of the 15 cases or five per cent of the total number.

Diatomaceous Earth

An examination of 108 men engaged in quarrying diatomaceous earth in California for use in fire brick, abrasives, concrete, etc., was reported by Legge and Rosencrantz[57] in 1932. This material contains 85 per cent free silica chiefly in the amorphous form. They found moderately advanced silicosis in 15 per cent of the men; advanced in six.

Fullers' Earth

McNally and Trostler[57a] reported finding severe pneumoconiosis in a group of men exposed to fullers earth. The material involved was chiefly montmorillonite, a complex magnesium calcium aluminum silicate.

Foundries

A study of 1,614 foundry workers was made by the Special Industrial Disease Commission of Massachusetts[58] in 1933. Silicosis was found in 8.8 per cent, silicosis with tuberculosis in 2.6 per cent, and tuberculosis alone in 0.9 per cent. Dust counts in the various departments were made and ranged from one to 266 million particles per cubic foot.

In 1934 McConnell and Fehnel[59] reported an examination of 215 foundry workers in 41 foundries. Sixty-seven, or 31 per cent, were diagnosed as showing X-ray changes indicative of early sili-

cosis. Dust counts and particle size determinations were made. The report discusses the high death rates among foundry workers for all respiratory diseases, brought out by the report of the Committee on Group Mortality which analyzed records of six large American and Canadian insurance companies.

In 1935 Warfield[60] reported the examination of 691 foundry workers of whom 129, or 18 per cent, showed silicosis.

In an investigation of 311 foundries in New York State in 1937, Greenburg, Siegal and Smith[85] made X-ray examinations of 4,066 foundry workers in 80 foundries and found that of these 2.7 per cent showed silicosis.

Other studies in this industry were reported in 1937 by Kelly and Hall,[60a] Osmond[60b] and Sander,[60c] in which the rates for silicosis were found to be 0.99, 5.5 and 7.0 per cent respectively.

In 1938 Trice and Easom[60d] reported the results of a study of 546 foundry workers in North Carolina. Only 1.5 per cent were found to have silicosis and no case had progressed beyond first stage. This low incidence was attributed to the fact that in this state foundry operations are conducted in open buildings.

An incidence for silicosis of 2.4 per cent was reported among 454 naval foundrymen by Brown and Klein[60e] in 1942, the median exposure being 29 years.

Granite Cutting

In a statistical study of the granite industry about Barre, Vermont, published in 1922, Hoffman[61] found that the mortality from pulmonary tuberculosis among granite cutters had increased from 257.7 per 100,000 in 1896 to 953.4 in 1918, while at the same time the tuberculosis mortality rate among the general population declined from 207.5 per 100,000 in 1896 to 96.4 in 1917. This excess in death rate was most marked among the men employed in cutting and especially among those who worked with pneumatic tools.

Such striking evidence of the apparent harmfulness of granite cutting induced the Public Health Service[62] to undertake an investigation at Barre lasting from 1924 to 1926 as part of a program aimed to cover the entire field of the dusty trades. Sanitary surveys of plants were carried out, including dust determinations. Sickness and mortality records were studied and physical examinations, including X-rays, made of a number of men. The percentage of free silica in the granite of that locality was found to vary between 31.8 and 38.6. The dust counts varied from 59.2 million particles per cubic foot, for hand pneumatic tool operators, to 1.9 for office employees.

Practically all granite workers were found to have early silicosis after four years employment and well advanced silicosis in 10 years. The general incidence rate of tuberculosis, including early cases was 6.5 per cent, while the death rate from tuberculosis alone was 14.1 per 1,000, a rate which is in excess of the death

rate for all causes in the general population. Among the facts noted in this careful and systematic investigation were the following: Universal occurrence of silicosis among the granite workers; a close relation between the intensity of dust exposure and the general health of the men; a sharp correlation between the length of exposure to dust and the prevalence of tuberculosis and the death rate from the disease; the death of a large proportion of workers from tuberculosis which required 20 or more years exposure to develop but was almost invariably fatal within a short time of onset, and the failure of workers to recover from their condition on going into non-dusty trades.

The conclusion was reached that the rising sickness and mortality rates from tuberculosis in this industry are due to longer use of the hand pneumatic tool.

The Special Industrial Disease Commission of Massachusetts[58] examined a group of 961 granite cutters in that State (1933) and found that 138, (14 per cent,) had silicosis and 73, (7.6 per cent,) had silico-tuberculosis. Thirteen, or 1.4 per cent, had tuberculosis only. Distribution of cases according to dust count groups is shown in the report.

In a study of 125 granite cutters from shops about New York City conducted by the Division of Industrial Hygiene and the New York Tuberculosis and Health Association,[63] 62 per cent showed silicosis. Tuberculosis definite or suspected (by X-ray) was present in 31 cases.

The Health Department of Virginia in 1938 reported that of 45 men employed in granite cutting, 12 showed "some degree of lung involvement", three with infection.[63a]

Granite Quarrying

Granite quarrying was investigated by the Public Health Service[64] in 1924–26. According to the report of this study the men were exposed to dust with a quartz content of 35.2 per cent. Seventy-five per cent of the particles were less than two microns in average diameter. Dust counts revealed very high concentrations in quarry hole drilling, 144.4 and 112.1 million particles per cubic foot respectively. Only the drillers showed pathologic lung changes. Of 36 drillers examined, half with an exposure of five to 19 years had silicosis, and four of the five who had spent more than 20 years at such work.

Grinding

The dust hazard in ax-grinding was investigated by Winslow and Greenburg[7] in a large plant in Connecticut in 1920. No physical examinations were made, but the mortality rates among the workers were studied and found to show a high incidence of tuberculosis which was attributed to wet grinding on sandstone wheels. This was shown to be far more hazardous by actual

dust count than dry grinding with an efficient exhaust. Dust counts in dry grinding shops with an exhaust system, for instance, showed an average of only one-twelfth as many particles per cubic foot as the average in the wet grinding shops.

Metal Polishing

A study of the health of workers in a *silverware* manufacturing plant was reported by the U. S. Public Health Service[65] in 1933. Dust counts in general showed less than five million particles per cubic foot. Pumice, emery, tripoli, sandstone, metal and rouge were the substances used. Of 51 X-rays taken none showed silicosis. Thirty-six were classified as having "more fibrosis than usual."

Of 80 metal polishers in various plants throughout New York City studied by the Division of Industrial Hygiene of the New York State Labor Department and the New York City Health Department,[66] two showed silicosis and 47 increased fibrosis.

Mining

Lead and Zinc—The first investigation of silicosis in mining in this country was made in 1914 and 1915 by A. J. Lanza and Edwin Higgins[67] of the U. S. Public Health Service and Bureau of Mines respectively among the lead and zinc miners of Joplin, Missouri. In that area the chief geologic formation is chert, a rock having a high percentage of free silica. Seven hundred miners were examined and of these 45 per cent were found to have silicosis, while 14 per cent had silicosis and tuberculosis and five per cent had tuberculosis. A further report of this same investigation was made by Lanza and Childs[68] containing the first detailed X-ray studies of silicosis made in this country.

Of the total 727 non-ferrous metal mine workers in three Utah mines examined by the United States Public Health Service,[67a] 9.1 per cent showed silicosis. Among the men with 20 or more years exposure to concentrations of 24 million particles (per cubic foot) or more, 68 per cent were so affected.

The Bureau of Mines[70] continued its investigations with a study of 309 men in the zinc mining district at Picher, Oklahoma, in 1923. Of these 30 per cent had silicosis. Finally in 1927 a permanent clinic for the study of the disease was established by the Bureau of Mines and the Metropolitan Life Insurance Co. at Picher.

Copper—Further investigations of mining conditions were made by Harrington and Lanza[69] in copper mines in Butte, Montana, in 1921. There, of 1,018 miners, 42 per cent showed definite signs of lung damage due to dust and six per cent were tuberculous.

In 1942 Ellis, Smith, Bonebrake and Hunter[70a] reported the result of a study of 6,243 miners in the Coeur D'Alene district of Idaho. Thirty-five per cent of those exposed from 11 to 19 years showed silicosis.

Coal—There is a prevalent opinion that coal miners do not suffer from silicosis or tuberculosis. But coal mining may produce silicosis when the associated rock has a sufficiently high silica content.

In 1935 the U. S. Public Health Service[71] published a report of the examination of 2,711 active workers in the *anthracite coal industry*. Of these, 616, or 22.7 per cent, showed silicosis of a slowly developing non-disabling type. After 25 years of exposure 90 per cent of the exposed had acquired the disease. Clinical pulmonary tuberculosis was found in 15 per cent of the early cases and in 43 per cent of the late ones. The percentage of free silica in the dust breathed by these men varied from 3–4 per cent for the regular miners to 35 per cent for the rock tunnellers and muckers. Dust counts varied from less than five to more than 300 million particles per cubic foot.

Clarke and Moffet[71a] found silicosis in 1 per cent of a group of 774 *soft coal miners* of the Southern Appalachian region.

Potteries

X-ray examination of 58 men engaged in making of bathroom fixtures was reported by Quaintance[72] in 1934. They were exposed to the dust of clay containing 20 to 25 per cent free silica. Five, or 8.6 per cent, were found to have third stage silicosis after an average exposure of 16 years.

In 1939 the United States Public Health Service[72a] reported an examination of 2,516 pottery workers in 9 factories in West Virginia. The quartz content of the dust ranged from 20 to 30 per cent. Six workers were found with third stage, 60 with second and 123 with first stage silicosis. Active and arrested cases of tuberculosis were three times as prevalent among potters with silicosis as among those without.

Rock Drilling, Blasting and Excavating

The apparent similarity of the risk in mining and the processes involved in excavating and tunnelling, so far as the silicosis hazard is concerned, led to an investigation of the latter processes in New York City by Smith and Fehnel,[73] in 1929. Analyses of specimens of rock drilled from various sites on Manhattan Island showed that the free silica content ranged from zero to 84 per cent. In the case of dry jackhammer drilling, 50 per cent of the silica particles were less than three to four microns in size, while with the water Leyner (wet) drill, 50 per cent of the particles were less than 1–8 microns in diameter.

Of the 208 drillers, blasters and excavators examined, 42 per cent showed early, and 15 per cent well-developed silicosis. Evidence of tuberculosis, including both active and inactive cases, was present in nine per cent of the total number.

Sand Blasting

The application of sand under air pressure to surfaces to be cleaned, freed from paint or etched, presents unquestionably one of the most serious silicosis hazards in industry today. Sand is always very high in free silica content, usually over 90 per cent, and it splits up on impact with the surface to which it is applied so that the dust particles become very small.

Greenburg and Winslow[74] in 1919 investigated conditions in the abrasive industry and made certain recommendations, and in 1932[75] they again reported a careful study of conditions in 28 metal working shops in four different states, using a total of 194 pieces of air blasting equipment. They found that when the sand blaster must work in the midst of his blasting he is exposed to air containing from 232 to 3,104 million particles per cubic foot, or from 23 to 310 times the amount of dust which is at present considered safe. Even when sand blasting is conducted in closed devices, the air of the general workroom adjacent to the device contains, they found, on an average, over 20 million particles of dust per cubic foot.

Sandstone Quarrying

At Amherst, Ohio, the sandstone centre of the country, Kindel and Hayhurst[76] examined 919 quarrymen in 1926. Of these, 30 per cent showed evidence of silicosis, advanced in seven per cent, while only two per cent showed silicosis with tuberculosis. The low incidence of tuberculosis was thought by the authors to be due possibly to an admixture of clay with the sandstone whose own free silica content varies from 94 to 97 per cent. Most hazardous was the job of grindstone turning where 77 per cent of workers were silicotic.

Slate Milling

Slate milling was studied by the U. S. Public Health Service[77] in New York State. The dust count in mining and milling operations was very high, 52 to 1,440 million particles per cubic foot. Eighty-seven per cent of 79 slate millers showed lung changes, but they were slight in all but four.

Spray Coating

The danger of silicosis resulting from the use of vitreous enamels in the manufacture of sanitary and other ware was investigated by the National Safety Council[78] and reported upon in the Final Report of its Spray Coating Committee, published in 1927. It was found that a real hazard may occur if ventilation is inadequate. Enamels examined by them which were used for spraying castings were found to contain from 21 to 37 per cent silica; those used for spraying sheet metal contained from 43 to 47 per cent silica.

Dust counts at the working face of spray coating booths ranged from 400,000 particles per cubic foot where the air velocity was 212 linear feet per minute (a low count,) to the excessive count of 445,000,000 particles per cubic foot where the exhaust ventilation was so poor as to be inappreciable. X-rays were obtained of nine workers who had been employed at this work for more than three years and of these, two showed definite and a third possible, silicosis.

Talc Mining

Ninety-three per cent of 57 talc miners were found to show slight lung changes in the study by the U. S. Public Health Service[77] reported in 1933.

In another study of the effect of talc made in two Georgia talc mills by the same agency[79] reported in 1935, 66 workers were divided into three groups according to dust exposure. The first group of 33 was exposed to 300 or more million particles of dust per cubic foot; the second of 13, to an average of 135 million particles and the third of 20, to an average of 17. Sixteen, or approximately half, of the mill workers in the first group showed nodular fibrosis of the lungs, equally divided between early and advanced stages. In the second group six of the 13 showed early changes. There were no changes in the third group.

The type of the talc in the region studied was hydrous magnesium silicate, $H_2Mg_3(SiO_3)_4$.

In 1943, Siegal, Smith and Greenburg[53c] reported a study of 221 tremolite (calcium magnesium silicate) talc miners and millers. Approximately 30 per cent of those exposed over 10 years showed a marked fibrosis which tended to be disabling in character. Calcifications of pleura, diaphragm and pericardium were present in 6.3 per cent of workers examined.

Trap Rock Quarrying

The results of an X-ray examination of 607 trap rock quarry workers were reported by Goldwater.[80] This rock has a low percentage of free silica, usually less than five. Of the entire group, two showed early and five more advanced silicosis, one complicated by tuberculosis.

CHAPTER V

PREVENTION OF SILICOSIS

The first step to be taken in the prevention of silicosis in any plant is an investigation of the existence and severity of the hazard. In other words, the free silica content of the dust in question, if not known, must be determined by suitable analysis, and its amount or intensity measured by dust counts.

Analysis of Dust for Free Silica

The principal methods for the determination of free silica are chemical, petrographic, x-ray diffraction and thermal methods. Various combinations of these methods are used, the most common of which is combined chemical and petrographic analysis.

Determining the presence of free silica in any material is a matter for an experienced investigator trained in chemistry, and if possible in petrographic analysis. Chemical methods alone can yield directly only the amount of total, or free and combined silica in a sample. From this figure the free silica must be calculated indirectly by ascertaining the amount in combination with bases known to be present and deducting this from the total silica. In dust containing a variety of minerals, it may be difficult to calculate the amount of uncombined silica correctly.

Drinker and Hatch[18] describe two methods for the determination of free silica and reference is made to their text book for details. The methods are (1) the combined chemical and petrographic method according to Knopf, and (2) the immersion method according to Ross and Sehl. Their studies show that in case of mixed dusts *it is necessary to separate the sample into size fractions and determine the composition of each fraction* in order to determine the true hygienic significance of the dust. For instance, the percentage by weight of free silica in the total sample of a foundry dust examined was 58.5 per cent, but the amount of free silica with a particle size less than 10 microns was only 5.7 per cent by weight of the total sample.

Dust Sampling Methods

Having determined the percentage of free silica in the dust in question and found it to be such as to present a possible silicosis hazard the next step is to determine the intensity of exposure by actually counting the number of particles in a given sample of air.

There are a number of methods of collecting dust samples, depending upon six general principals of operation, namely: Settlement, filtration, washing, impingement, electric precipitation and thermal precipitation. Those best known are the Kotze Konimeter, which has been employed in routine dust sampling in South Africa for

more than 20 years; the Owens Jet Dust Counter which was long popular in Great Britain, and the Greenburg-Smith Impinger, the official instrument of the U. S. Public Health Service. All of these employ the principle of impingement.

Konimeter

The Konimeter is a small instrument which can be held in the hand and has the advantages of simplicity and ease of operation. Air is drawn in through a nozzle by means of a spring-actuated pump and impinges upon a collecting plate. Samples of 2.5 and 5 cc. of air (and multiples of these) may be collected. The glass plate upon which the air impinges is covered with a thin film of petroleum or glycerin jelly which retains the dust. Thirty samples can be collected on a single disc. Immediate examination of the spots under the microscope follows the collection of the samples.

The disadvantages of the Konimeter are that the dust-collecting efficiency is low, and it is selective in respect to size of particles.

Owen Jet Dust Counter

The Owen Jet Dust Counter is somewhat similar to the Konimeter but it has a capacity of 50 cc. and the impinging surface is an ordinary cover slip. It has essentially the same advantages and disadvantages of the former.

Greenburg-Smith Impinger

With the Greenburg-Smith Impinger apparatus a known amount of air is drawn through a tube into a flask of distilled water. As the air leaves the tube it strikes a glass impinging plate submerged beneath the surface of the water. The dust particles in the air are thus caught in the water, diluted and placed in a counting chamber for microscopical examination and counting.

Electric Precipitator

This is an instrument which, though less used than the other three in the past, has a high degree of efficiency and is used by the U. S. Bureau of Mines in testing respirators. Dust laden air is passed between two surfaces carrying a high electric potential and under the force of the electric field the particles are driven in a direction normal to the air motion and so precipitated upon the collecting surface. Samples cannot be examined by direct microscopic count but must be washed out into water or other liquid first.

Thermal Precipitator

This instrument is popular in Great Britain at the present time but is not used to any extent in this country. It depends upon the principle that a dust free area will occur around a hot rod.

Dusty air is drawn through a slot across which is placed an electrically heated wire. The walls of the slot are formed by cover slips kept cool by a backing of brass blocks which act as heat conductors. The dust is deposited on the cover glasses which can be removed for counting.

For a complete description of the above methods, and others, the reader is referred to the chapter on "Dust Concentration" in Industrial Dust by Drinker and Hatch.[18]

Figure 5

Chemist of Division of Industrial Hygiene, New York State Department of Labor, Taking Sample for a Dust Count

Particle Size Distribution

In addition to counting the number of particles in a given sample, an estimate of particle size distribution—that is to say the percentage of particles of various sizes under 10 microns—is valuable. With the Owens apparatus it is possible to measure particles as small as 0.5 microns in diameter while even smaller particles can be distinguished and their presence recorded. Particles of one micron in size are shown by the Greenburg-Smith Impinger method and an experienced observer can detect particles as small as 0.7 microns. In a study of 26 samples of various industrial dusts Bloomfield found that most of the particles—69 per cent on an average—were between one and three microns in average diameter. Twenty-one per cent were less than one micron and two per cent were less than 0.5 microns.

Hatch and Pool[81] advocate the use of dark field illumination with a special counting cell to obtain greater magnification. With the dark-field microscope, particles down to slightly more than 0.1 micron in size can be enumerated.

Preventive Measures

When an injurious amount of an injurious dust has been demonstrated by dust analysis and dust counts, the question of how to correct the situation next arises.

The prevention of silicosis involves several different angles of attack depending upon the particular problem under consideration, and usually several methods in combination are necessary for the best results. The methods of attack all follow certain familiar principles. They may be divided logically into three groups: Those having to do with the process; those having to do with the work place, and those having to do with the worker himself.

Related to Process

The first group of preventive measures, those related to the process, comprises the following:

1. Substitution of non-silicosis-producing material.
2. Enclosure and segregation of dusty processes.
3. Local exhaust ventilation for the removal of dust at point of origin.
4. Suppression of dust by water.

Related to Work Place

The second group of preventive measures, those related to the work place, includes:

5. General artificial ventilation.
6. Plant cleanliness.

Related to Worker

The third group, relating to the worker, comprises:

7. Direct protection of the worker.
8. Alteration of work.
9. Medical supervision.
10. Education.
11. Use of aluminum powder.

Substitution

This solution of the problem of prevention is at once the simplest, where practicable, and the most satisfactory. It has been applied successfully in several important industries.

In abrasive blasting the use of a metal abrasive instead of sand reduces the free silica content of the dust to less than five per cent from 42 per cent or above. Metal abrasive, steel grit, lasts longer than sand and is consequently cheaper. It can be used with the same equipment. Aluminum oxide also makes a satisfactory substitute for sand, especially in monument work, and has the advantage of greater cutting power.

The widespread use of artificial stones of silicon carbide or aluminum oxide instead of natural stones in metal grinding is another example of substitution with very beneficial results so far as silicosis is concerned. Studies by Clark[83] have shown no silicosis hazard in exposure to the dust of aluminum oxide, and no increased evidence of tuberculosis.

The silicosis hazard which exists in foundries, is due to sand blasting of castings and also the use of "parting compounds" for dusting molds which are often very high in free silica content. In addition to substitutes for sand in blasting, non-siliceous parting compounds are available and are quite generally being substituted.

The pottery industry, especially abroad, has always been associated with high silicosis rates due to the use of flint in bedding the ware for firing. Some plants have reduced this hazard markedly by the substitution of clay with its low free silica content for flint which is pure quartz.

These are a few outstanding examples of silicosis prevention by substitution. Other applications of the principle may be anticipated in the future. The first thing for the prevention minded employer to ask himself is: "Do I need to use this dangerous material?"

Isolation of Dusty Processes

Next to removing the silicosis hazard entirely by giving up the use of siliceous material, possibly the most effective safeguard when practicable, is isolation of the dusty process either by mechanical enclosure or geographical segregation.

Some processes, notably certain types of sandblasting, lend themselves admirably to enclosure by barrels, tables or cabinets which can be constructed so that very little dust escapes into the workroom. Bloomfield and Greenburg[84] found in a study of enclosed sandblasting operations, that with proper apparatus properly maintained the average amount of dust in rooms at large was less than five million particles per cubic foot. Strict care in upkeep is necessary, however, for under less than ideal conditions the average room atmosphere where this type of work was carried on contained over 20 million particles of dust per cubic foot, a dangerous concentration.

Aside from enclosure in mechanical systems, situations occur where dusty processes can be isolated in such a way that a minimum number of workers will be exposed. Measures for the control of silicosis in metal grinding in Canada provide for the "racing" of grindstones in separate rooms. Surfacing machines in stone yards could be similarly isolated. Instead of this, they are often operated in the midst of the yard where the dust created affects everybody present.

Isolation of dusty processes by *time*, as well as geographically, is another possibility under some circumstances. An example is the shake-out process in foundry work which is often done at night when only the shake-out crew itself is exposed.

Figure 6

Rock Drilling—No Dust Control—Serious Silicosis Hazard

Local Exhaust Ventilation

For the many dusty processes where silica is necessarily present and isolation is not practicable, the best preventive measure is removal of dust at point of origin by exhaust ventilation. There is almost no limit to the possible application of this principle. It is widely used in such operations as grinding, polishing, mixing, filling, etc., and new applications are continually being developed. One of the most important and recent of these is a method of exhaust for the removal of the dust in rock drilling, long a very serious source of silicosis in mines and tunnel construction. Another hazardous trade where control is dependent upon properly applied local exhaust ventilation is stone cutting.

Figure 7

Rock Drilling—Dust Removal by Vacuum Method—Silicosis Hazard under Control

There is no need here to discuss details of such ventilation since each situation presents its own particular problems in the engineering field. The crucial test of whether or not a system is adequate, lies in its ability to maintain the dust in the air at a low level and its efficacy should always, therefore, be tested by dust counts.

Water

Suppression of dust by the use of water as a spray or stream applied at the point of origin of the dust has a place in the control of silicosis but its value should not be overestimated. The finest particles are not laid by water but actually held in suspension in the air by droplets of moisture. Dry drilling under adequate exhaust will produce lower dust counts than wet drilling. Winslow and Greenburg,[7] in their investigation of an ax factory found much more dust present under conditions of wet than dry grinding when the latter was done with proper exhaust. With wet methods, the dampened dust itself must be removed or it will dry out and constitute a hazard all over again.

Under some circumstances, wet methods especially in combination which general ventilation are valuable. Reduction of dust and consequently the silicosis rate in the South African mines has been accomplished through these means. In operations, such as stone crushing and grinding, dust may be reduced by the use of water when other measures are impracticable.

General Artificial Ventilation

Changing the air of the workplace frequently by artificial ventilation, bringing in fresh air and removing the stale is a valuable adjunct to other methods of control. Its effect is actually to dilute the dust present. In the mines already mentioned the use of wet drills is combined with general artificial ventilation by fans which ensure a circulation of about 60 cubic feet per person, per minute.

Plant Cleanliness

One of the most important parts in dust control is played by good housekeeping, that is to say by measures to promote plant cleanliness. In well kept foundries it is estimated that such measures account for 75 per cent of dust control. If a plant is not kept clean, dust accumulating from day to day, settling on floors and other parts of the workrooms, adds materially to the general hazard by being stirred up and re-circulated. When circumstances are appropriate, arrangements for vacuum cleaning or hosing down workrooms at intervals, preferably outside working hours, should be included as a matter of course in all silicosis prevention programs. Special arrangements for the care of dusty material come under this heading. A case in point is the use of a grating in the floor of a sandblasting room to allow the sand to fall through to a container instead of accumulating on the floor. Under this item of good housekeeping, too, should be included the careful upkeep of all ventilating devices and other protective apparatus. If this is not done even the best equipment will deteriorate rapidly.

Masks and Helmets

Direct protection of the worker by the use of masks or helmets is impracticable in many situations owing to the difficulty of a man's carrying on heavy work when so encumbered; so that in general, control should be sought by other means, but there are situations when this form of protection is satisfactory and even necessary. In abrasive blasting, for instance, when the operator must be in the same room with the object being cleaned, a positive pressure helmet, that is to say a helmet in which pure air is provided from an outside source, is absolutely essential in addition to room ventilation. Various types of helmets of satisfactory design are available. In studies made by Bloomfield and Greenburg[84] it was found that with a properly made helmet an air flow into the helmet of six cubic feet per minute was sufficient to supply clean air (dust count under three million particles per cubic foot) under all conditions encountered. It is of course essential that the helmet be inspected frequently for defects and kept in sound condition. Incidentally the same studies revealed the total inadequacy of non-positive-pressure helmets in abrasive blasting. Where no outside air was supplied the dust counts under these helmets averaged 581 million particles per cubic foot. In such circumstances the helmet gives a totally false sense of security.

Masks represent a cheap and simple type of direct protection which has much in its favor under appropriate conditions. They are eminently adapted to a situation where the dust exposure is moderately severe but of short duration. Masks of high efficiency against silica-containing dust have been developed which are at the same time reasonably comfortable and offer relatively little resistance to breathing. If such masks are properly maintained, there can be no objection to them and they might under such circumstances, take the place of expensive ventilating equipment. But unfortunately the human element, ignorance or indifference on the part of the worker and laxity on the part of the employer, combine to destroy their effectiveness. It is the common carelessness found in their use, together with the sense of false security when carelessness exists, which renders them unsatisfactory as a rule in actual practice.

Medical Supervision

In enacting its so-called "silicosis legislation" in 1936, the New York Legislature said:

"It is hereby declared to be the policy of the Legislature of this State, in enacting this Article, to prohibit through every lawful means available, any requirement as a pre-requisite to employment which compels an applicant for employment in any occupation coming within the purview of this Article to undergo a medical examination."

This statement of policy is designed to protect the worker's right to a job. As one labor representative expressed it: "We want our jobs whether we have silicosis or not".

However, the young worker entering a dusty industry for the first time will be doing himself a distinct service if he subjects himself to a medical examination to determine that he is not peculiarly susceptible to dust irritation. Such a worker should repeat these examinations at regular intervals to keep a constant check-up on his condition.

Such a procedure will absolutely protect the individual worker from silicosis if he heeds the information disclosed by these examinations. First, because it is known that people who have had previous lung disease or who have any obstruction to breathing will get the disease more easily than others and hence should not be employed for this work for their own good. Secondly, it is necessary to know whether measures in use are in fact effective, and this can only be ascertained by periodic check-up examinations to discover whether silicosis is developing. Thirdly, if early presilicotic changes in the lungs are found by X-ray examination following dust exposure, silicosis may be avoided by prompt action in changing to another type of work.

The interval between examinations should depend upon the intensity of exposure. Where it is moderate, annual check-ups will probably be sufficient; where very severe, re-examinations had best be made at least every six months. The initial examination should include a complete physical survey with an X-ray of the chest. Subsequent check-ups may be limited to chest X-ray examinations only.

Education

Education of the worker in the nature of the risk he is running, the part he can play in preventing the disease by intelligent use of preventive equipment and the value to himself of physical examinations should be a part of every prevention program. If more attention were paid to education the advantage of physical examinations as a device to keep men healthy and preserve their working capacity would be apparent.

Use of Aluminum

A development of promise in the field of silicosis prevention was the discovery by Denny, Robson and Irwin[85a] that silicotic fibrosis could be completely prevented experimentally in rabbits exposed to high concentrations of quartz dust if one per cent or more of metallic aluminum were added. The authors believe this effect is due to the continous formation of aluminum hydroxide which acts by forming an insoluble and impermeable coating on the quartz grains, thus preventing their solubility in lung tissues. Gardner

has found that amorphous hydrated alumina in powder form is also effective.

Inhalation of aluminum powder for the prevention and treatment of silicosis is now under trial in a number of mines and other dusty industries in this country and Canada. As yet insufficient data has been accumulated to asses its value properly. Whatever this may prove to be it is obvious that it should not be resorted to except as an adjuvant to suitable measures of engineering control.

Preventive measures have been discussed separately but in practice, no one alone, except of course the first—substitution of a harmless substance—is enough, and the more that are used in combination the more complete will protection be. A satisfactory and comprehensive program for the prevention of silicosis in the majority of dusty industries will include physical supervision and education, alternation of men at dusty and non-dusty work, segregation of dusty processes, local exhaust ventilation, or masks or helmets, or both and finally plant cleanliness and an invincible determination on the part of those responsible to keep down dust. The efficiency of measures to remove or control dust can be demonstrated quite simply by dust counts and these should always be made at intervals in conjunction with a preventive program.

New York State Codes for the Control of Dust

Action on the part of State Departments to formulate rules for controlling dust in specific industries is a highly constructive approach to the problem of silicosis prevention.

New York State, in Industrial Code Bulletins Nos. 33, 34 and 35, has formulated Codes for dust control in rock drilling, stone crushing, and stone cutting and finishing, respectively.

In *rock drilling* and *stone crushing*, maximum allowable dust concentrations are as follows:

Class of Stone	Free Silica Dioxide Content of Stone	Maximum Allowable Atmospheric Dust Concentration
I	Any stone formation having free silicon dioxide as a component part and containing uniformly less than ten (10) percent by weight of free silicon dioxide.	100,000,000 particles per cubic foot of air.
II	Any stone formation having free silicon dioxide as a component part and containing ten (10) percent or more by weight of free silicon dioxide.	10,000,000 particles per cubic foot of air.

In Code Bulletin No. 35 relating to *stone cutting* and *finishing*, a third class of stone is recognized containing 70 per cent or more by weight of free silicon dioxide. When handling stone of

this character, the atmospheric dust concentration must be limited to 5,000,000 particles per cubic foot of air.

Control of the dust hazard in the construction of public works is also required in New York State according to Section 222a of the Labor Law which reads as follows:

In the construction of public works by the State or a public benefit corporation or a municipal corporation or a commission appointed pursuant to law wherein a harmful dust hazard is created for which appliances or methods for the elimination of harmful dust have been approved by the Board of Standards and Appeals, a provision shall be inserted in each contract for the construction of such work requiring the installation, maintenance and effective operation of such appliances and methods, and a further provision shall be inserted in such contract that if this Section is not complied with, the contract shall be void. In the construction of public works performed directly by the State or a public benefit corporation or a municipal corporation or a commission appointed by law, wherein a harmful dust hazard is created for which appliances or methods for the elimination of silica dust or other harmful dust have been approved by the Board of Standards and Appeals, the department, board or officer in the State, public benefit corporation, or municipal corporation or commission or board appointed pursuant to law, having jurisdiction over the construction of such work shall provide for the effective use of such approved appliances or methods in connection therewith. A violation of this Section shall constitute a misdemeanor and shall be punishable by a fine of not more than $500 or by imprisonment for not more than one year or by both fine and imprisonment.

CHAPTER VI

COMPENSATION FOR SILICOSIS

Compensation for Silicosis in New York State

Unquestionably the single most potent factor in silicosis prevention is the making of silicosis a compensable disease. Efforts to do this in New York State were finally successful in 1936.

Sections of the Law dealing with compensation for silicosis in this State are given below:

WORKMEN'S COMPENSATION LAW—ARTICLE 4-A

Silicosis, and Other Dust Diseases

65. **Prevention of Silicosis and Other Dust Diseases.** 1. It is hereby declared to be the policy of the Legislature of this State, in enacting this Article, to prohibit through every lawful means available, any requirement as a pre-requisite to employment which compels an applicant for employment in any occupation coming within the purview of this Article to undergo a medical examination.

2. The board of standards and appeals is hereby required to add to the industrial code, as provided in sections twenty-eight and twenty-nine of the labor law, effective rules and regulations governing the installation, maintenance and effective operation in all industries and operations wherein silica dust or other harmful dust hazard is present, of approved devices designed to eliminate such harmful dusts and to promulgate such other regulations as will effectively control the incidence of silicosis and similar diseases.

66. **Compensation Payable for Disability or Death.** Compensation shall not be payable for partial disability due to silicosis or other dust disease. In the event of temporary or permanent total disability or death from silicosis or other dust disease, notwithstanding any other provision of this Chapter, compensation shall be payable under this Article to employees in the employments enumerated in Section 3 of this Chapter or to their dependents in the following manner and amounts: If disablement or death occur during June, nineteen hundred thirty-six, not exceeding the sum of five hundred dollars; thereafter the total of compensation and benefits payable for disability and death shall increase at the rate of fifty dollars each calendar month until and including the month of December, nineteen hundred forty-three. The aggregate amount payable shall be determined by the total amount payable in the month in which disablement or death occurs. In no event shall such compensation exceed an aggregate total of five thousand dollars for temporary total disability and six thousand five hundred dollars for permanent total disability or death.

Compensation payable hereunder shall be paid from the eighth day following total disablement at the rate of 66 and two-thirds per centum of the average weekly wage to be computed under Section 14 of this Chapter; but in no case shall compensation exceed $25 per week nor in the event

of total disability be less than eight dollars per week; provided, however, that in the event of death from such disease his dependents shall receive, in the manner provided by Sections 16 and 17 of this Chapter, any balance remaining between the amounts paid for disability and the total compensation payable under this article.

Notwithstanding the provisions of Section 28 of this Chapter, all claims for compensation resulting from inhalation of harmful dust, where the last exposure occurred between September first, nineteen hundred thirty-five and June sixth, nineteen hundred thirty-six, shall be barred unless filed within one hundred and eighty days from June sixth, nineteen hundred thirty-six.

67. **Liability of Employer.** An employer shall be liable for the payments prescribed by this article for silicosis or other dust disease when disability of an employee resulting in loss of earnings shall be due to an employment in a hazardous occupation in which he was employed, and such disability results within one year after the last injurious exposure in such employment; or, in case of death resulting from such exposure, if such death occurs within five years following continuous disability from such disease. The provisions of section forty-four of this chapter shall not apply to claims arising under this article.

The employer in whose employment the employee was last injuriously exposed in a hazardous occupation and the insurance carrier, if any, which was on the risk at the time of the last injurious exposure in such employment, shall be liable for any payments required by this article; the notice of injury and claim shall be made to such employer.

Any exposure to the hazards of harmful dust in this state for a period of sixty days after September first, nineteen hundred thirty-five, shall be presumed, in the absence of substantial evidence to the contrary, to be an injurious exposure.

68. **Medical Treatment and Hospital Care.** Notwithstanding any other provisions of this chapter the medical treatment herein provided for or, in lieu thereof, such hospitalization as the board may allow, shall be limited in the case of an employee disabled by an occupational disease due to or resulting from the inhalation of harmful dust to a period of ninety days from the date of such disablement, but the requirement for such medical treatment or hospitalization may be extended for an additional period, not necessarily continuous, not to exceed three hundred and sixty days upon the order of the industrial board.

In determining the medical treatment, hospitalization, and other care required beyond the period of ninety days from the date of disablement, the board shall consider the recommendations contained in the report submitted by the committee of expert consultants as required under the provisions of section seventy-one of this chapter.

Copies of the order of the board directing the claimant as to the proper type of treatment, hospitalization and other care to be secured shall be sent to all parties in interest and also to the attending physician and medical director of any hospital, sanatorium or other place in which the treatment or care is being given. No claim for such treatment or care not in accordance with the requirements of the order of the board shall be valid and enforceable for any period more than five days after such notice of direction and report shall have been sent.

69. **Workers, When Not Entitled.** If an employee, at the time of his employment, falsely represents in writing that he has not previously been disabled from the disease which is the cause of disability or death or has not received compensation or benefits under this article, no compensation shall be payable.

70. **Special Medical Examiners.** The industrial commissioner shall divide the state into five districts and in each district may appoint two or more special medical examiners who shall be licensed physicians in good professional standing, each of whom shall have had, at the time of his appoint-

ment, and immediately prior thereto, at least five years of practice in the diagnosis, care and treatment of pulmonary diseases. Such examiners shall be employed on a per diem basis as the exigencies of the work may require. Fees of examiners shall be fixed by the industrial commissioner within the limits of the appropriation therefor. Each position of special medical examiner provided herein shall be in the exempt class of civil service.

Whenever a claim is made under this article and an examination of the claimant by an impartial physician is desired by any party in interest, the industrial commissioner shall order such medical examiners to make the necessary medical and x-ray examination of the claimant in an effort to obtain the medical facts in an impartial manner.

For the purposes of adjudication under this chapter, the industrial board shall adopt rules of practice and procedure and shall prescribe methods and standards under which physical examinations, x-rays and other studies shall be conducted.

71. **Expert Consultants.** The industrial commissioner shall appoint as a committee of expert consultants on dust diseases three licensed physicians in good professional standing, each of whom shall have had, at the time of his appointment, and immediately prior thereto, at least ten years of practice in the diagnosis, care and treatment of diseases of the pulmonary tract, along with interpretation of x-ray films thereof. One of such committee shall be designated by the commissioner as chairman. They shall each be paid a salary of seven thousand five hundred dollars per year. Each such position of consultant shall be in the exempt class of civil service.

As soon as practicable after the filing of a claim for compensation hereunder, or notice thereof, the commissioner shall direct an examination of the claimant by the committee of expert consultants, or one of them, including such x-ray and other pathological examinations and tests as in their opinion may be necessary for the purpose of determining diagnosis, disablement, causal relation to the employment and the nature and type of medical treatment, hospitalization and other care required. In the event that the claim is not controverted as to any medical fact, the examination and report of one member of the committee of expert consultants shall be deemed the examination and report of the committee. In the event that the claim is controverted upon any medical ground, the report shall be made by the full committee after a physical examination by at least one such expert consultant. The findings and opinions of a majority of the committee of expert consultants shall constitute the findings and opinion of the committee. The contents of such report of the committee of expert consultants introduced in evidence shall constitute prima facie evidence of fact as to the matter contained therein, and any of the makers of such report shall be subject to examination upon demand.

Copies of the report shall be sent to all parties in interest and also to the attending physician and medical director of any hospital, sanatorium or other place in which treatment or care is being given.

Such expert consultants, or any of them, to assist in reaching a conclusion as to the care and treatment needed shall have the right to require the attending physician or medical director of any hospital or sanatorium or other place in which treatment or care provided for by this section is being given, to attend at such time or place as may be reasonably convenient, to consult with such expert consultants, or any of them, and to describe the nature and type of care or treatment being rendered, and for such attendance shall be entitled to receive a fee from the employer, or carrier, in an amount to be fixed by the commissioner in addition to any fee payable under section one hundred twenty.

In the event of a claim for death benefits, the committee of expert consultants upon their own initiative or upon the order of the commissioner or the board shall examine all available evidence pertaining to such claim, including medical and hospital records, x-rays and other reports made during the lifetime of the deceased, including the findings of any autopsy, and shall render its findings and report thereon.

The industrial commissioner or the industrial board shall on their own volition or on the application of either an employee, an employer, or an insurance carrier, direct such expert consultants to make examinations of claimants, to review the findings of special medical examiners, to read and review the files of compensation cases when necessary, and to inform the industrial commissioner and the industrial board of their opinion as to their findings in such cases.

Such committee of expert consultants shall as soon as practicable and at the direction of the industrial commissioner, make such investigation as shall be necessary for diagnosing and evaluating the progressive degrees of disability and incapacity that may result from silicosis or other dust diseases, and for determining the feasibility of allowing compensation for partial disability and other full benefits under this chapter in such cases.

72. **Alternative Remedy.** The liability of an employer prescribed by this article shall be exclusive and in place of any other liability whatsoever, at common law or otherwise, to such employee, his personal representatives, husband, parents, dependents or next of kin, or anyone otherwise entitled to recover damages, at common law, or otherwise on account of any injury, disability, or death, caused by the inhalation of harmful dust, except that if an employer fail to secure the payment of compensation for his injured employees and their dependents as provided in section fifty of this chapter, an injured employee, or his legal representative in case death results from the injury or disease, may, at his option, elect to claim compensation under this chapter, or to maintain an action in the courts for damages on account of such injury or disease; and in such an action it shall not be necessary to plead or prove freedom from contributory negligence nor may the defendant plead as a defense that the injury or disease was caused by the negligence of a fellow servant or that the employee assumed the risk of his employment, nor that the injury or disease was due to the contributory negligence of the employee.

Compensation for Silicosis in Other States

The rapidly developing interest in and knowledge of conditions affecting the health of workers has resulted in quite a marked recent increase in State laws providing for occupational disease.

Compensation for silicosis is* compulsory in the following States:

Arkansas	Massachusetts	Ohio
California	Michigan	Utah
Delaware	Minnesota	Washington
Idaho	New York	Wisconsin
Maryland	North Dakota	

Application of the law making silicosis compensable is elective in the following States:

Connecticut	Nebraska	Pennsylvania
Illinois	New Jersey	Rhode Island
Indiana	North Carolina	Virginia
Kentucky	Oregon	West Virginia
Missouri		

* As of December 1, 1944.

BIBLIOGRAPHY

1. Gardner, L. U.: Pathology of the pneumoconioses. N. Y. State Jour. Med., 36, 1377, 1936.
2. Lanza, A. J. and Vane, R. J.: The prevalence of silicosis in the general population and its effects upon the incidence of tuberculosis. Am. Rev. Tuberc., 29, 8, 1934.
3. Registrar—General's Decennial Supplement. England and Wales, 1931. Part 2A. Occupational Mortality, London, H. M. Stationery Office Pub. 1938.
4. Irvine, L. G., Simson, F. W. and Strachan, A. S.: The clinical pathology of silicosis. Silicosis. International Labor Office. Geneva, 1930.
5. Thompson, L. R. and Britten, R. H.: The silica dust hazard in the granite cutting industry. Jour. Ind. Hyg., 12, 123, 1930.
6. Dreessen, W. C. and Jones, R. R.: Anthracosilicosis. Jour. Am. Med. Assoc., 107, 1179, 1936.
7. Winslow, C. E. A. and Greenburg, L.: A study of the dust hazard in the wet and dry grinding shops of an ax factory. Pub. Health Repts., 35, 2393, 1920.

7b. Merewether, E. R. E.: A memorandum on asbestosis. Tubercle 15, 109, 1933; 152, 1934.

8. Collis, E. L. and Yule, G. U.: The mortality experience of an occupational group exposed to silica dust, compared with that of the general population and an occupational group exposed to dust not containing silica. Jour. Ind. Hyg., 15, 395, 1933.
9. Gardner, L. U.: Silicosis and related conditions. Jour. Ind. Hyg., 19, 111, 1937.
10. Audley, J. A.: Silica and the silicates. Bailliere, Tindall and Cox, London, 1921.
11. Mellor, J. W.: Modern inorganic chemistry. Longmans, Green & Co., New York, 1922.
12. Gregory, T. C.: The condensed chemical dictionary. Chemical Catalog Co., New York, 1930.
13. Jones, W. R.: Silicotic lungs, the minerals they contain. Jour. Hyg., 33, 307, 1933.
14. Gardner, L. U.: The experimental production of silicosis. Pub. Health Repts., 50, 695, 1935.
15. Ladoo, R. B.: Nonmetallic Minerals. Occurrence, Preparation, Utilization. McGraw-Hill Book Co. New York, 1925.
16. Silicosis. Records of the International Conference held at Johannesburg, August 1930. International Labor Office. Geneva, 1930.
17. Middleton, E. L.: Industrial pulmonary disease due to inhalation of dust. Lancet. 2, 1, 59, 1936.
18. Drinker, P. and Hatch, T.: Industrial Dust. McGraw-Hill Book Co. Inc. New York and London, 1936.

18a. National Silicosis Conference. Summary reports submitted to the Secretary of Labor by Conference Committees, February 3, 1937. Bulletin 13. United States Dep't. of Labor, Division of Labor Standards.

19. Gye, W. E. and Purdy, J. S.: The poisonous properties of colloidal silica; the effects of the parenteral administration of large doses. Brit. Jour. Exp. Path., 3, 75, 1922.
20. Mills, R. G.: The effect of prolonged exposure of the siliceous spicules of a fresh water sponge (spongilla fragilis) to the action of animal tissues. Am. Jour. Hyg., 13, 224, 1931.

21. Belt, T. H.: The pathology of silicosis of the lung. Can. Pub. Health Jour., 20, 495, 1929.

22. Heffernan, P. and Green, A. T.: The method of action of silica dust in the lung. Jour. Ind. Hyg., 10, 272, 1928.

23. Cummins, S. L. and Weatherall, C.: Effects of colloidal silica upon the growth of tubercle bacilli in blood. Brit. Jour. Exper. Path., 12, 245, 1931.

24. Gye, W. E. and Kettle, E. H.: Silicosis and Miners' phthisis. Brit. Jour. Exper. Path., 3, 241, 1922.

25. Gardner, L. U.: Will the inhalation of siliceous dusts activate a partially healed focus of tuberculous infection? Pub. Health Repts., 45, 282, 1930.

26. Dowd, G.: A bacteriological study of attenuated (R.) tubercle bacilli recovered from silicotic and normal guinea pigs. Am. Rev. Tuberc., 32, 62, 1935.

26a. Vorwald, A. J. and Delahant, A. B.: The influence of silica on the nature and acquired resistance to the tubercle bacillus. Am. Rev. Tuberc., 38, 347, 1938.

27. King, E. J. and Dolan, M.: Silicosis and metabolism of silica. Can. Med. Assoc. Jour., 31, 21, 1934.

28. Bloomfield, J. J. and Goldman, F. H.: The urinary excretion of silica by persons exposed to silica dust. Pub. Health Repts., 50, 421, 1935.

29. Goldwater, L. J.: The urinary excretion of silica in non-silicotic humans. Jour. Ind. Hyg., 18, 163, 1936.

30. Sweany, H. C., Porsche, J. D. and Douglass, J. R.: Chemical and pathological study of pneumoconiosis with special emphasis on silico-tuberculosis. Arch. Path., 22, 593, 1936.

30a. Oranguren, H. and Bartholin, E.: Contribution to the diagnosis of pulmonary silicosis by the investigation of silica in the blood and sputum. Rev. Med. De Chile, 71, 464, 1943.

31. Miller, J. W. and Sayers, R. R.: The physiological response of the peritoneal tissue to dusts introduced as foreign bodies. Pub. Health Repts., 49, 80, 1934.

32. McCord, C. P., Fleming, R. L., Ainslee, H. and Johnston, J.: The measurement of the harmfulness of dusts for humans through the agency of animal reactions. Surg. Gyn. and Obstet., 63, 129, 1936.

33. Stüber, K.: The cellular response of lymph nodes to various dust suspensions introduced into lymphatics. Jour. Ind. Hyg., 16, 282, 1934.

34. Sutherland-Strachan, A. and Simson, F. W.: A preliminary study of the pathology of silicosis as seen on the Witwatersrand. Silicosis, International Labor Office. Geneva, 1930.

35. Willis, H. S.: Pneumoconiosis and tuberculosis. Medicine, 9, 413, 1930.

36. McNally, W. D.: Silicon dioxide content of lungs in health and disease. Jour. Am. Med. Assn., 101, 584, 1933.

37. Pancoast, H. K. and Pendergrass, E. P.: A review of our present knowledge of pneumoconiosis based on roentgenological studies with notes on the pathology of the condition. Amer. Jour. Roentgenol., 14, 381, 1925.

38. Lehmann, G.: The significance of the filtering action of the nose on the incidence of pneumoconiosis. Med. Wchnschr., 80, 1166, 1933.

38a. Proske, H. O. and Sayers, R. R.: Pulmonary infection in pneumoconiosis. A bacteriologic and experimental study. Pub. Health Repts., 49, 839, 1934.

38b. Coggin, C. B., Griggs, D. E., and Stetson, W. L.: The heart in pneumoconiosis. Am. Heart J., 16, 411, 1938.

38c. Gardner, L. U.: The pathology and roentgenographic manifestations of pneumoconiosis. J. A. M. A., 114, 535, 1940.

39. Dible, J. H.: Silicosis and malignant disease. Lancet, 982, 1934.

40. Berblinger, W.: The increase of lung cancer and diseases of the lung caused by dust. Abst. Med. Klin., 27, 1337, 1931, Am. Jour. Can., 16, 839, 1932.

41. Teleky, L.: Occupational cancer of the lung. Jour. Ind. Hyg. and Tox., 19, 73, 1937.

42. Pancoast, H. K. and Pendergrass, E. P.: A review of pneumoconiosis; further roentgenological and pathological studies. Am. Jour. Roentgenol., 26, 574, 1931.

43. Gardner, L. U.: The diagnosis of silicosis with special reference to roentgenological manifestations. Ann. Int. Med., 10, 166, 1936.

44. Pancoast, H. K., Pendergrass, E. P., Riddell, A. R., Lanza, A. J., McConnell, W. J., Sayers, R. R., Sampson, H. L. and Gardner, L. U.: Roentgenological appearances in silicosis and the underlying pathological lesions. Report by Committee Composed of doctors. Pub. Health Repts., 50, 989, 1935.

45. Irwin, D. A.: Histological demonstration of siliceous material by microincineration. Canad. Med. Assoc. Jour., 31, 135, 1934.

46. Report of the Committee on Pneumoconiosis of the Am. Pub. Health Assoc. Am. Pub. Health Assoc. Year Book, 1932–33.

46a. Crombie, D. W., Blaidell, J. L. and MacPherson, G.: The treatment of silicosis by aluminum powder. Canad. M. A. J., 50, 318, 1944.

47. Kessler, H. H.: Silicosis in the abrasive powder industry. Am. Jour. Pub. Health, 21, 1930, 1931.

48. Chapman, E. W.: Acute silicosis. Jour. Am. Med. Assoc., 98, 1439, 1932.

49. Kilgore, E. S.: Pneumonoconiosis, an unusually acute form. Jour. Am. Med. Assoc., 99, 1414, 1932.

50. An investigation relating to health conditions of workers employed in the construction and maintenance of public utilities. Hearings before a subcommittee of the committee on labor. House of Representatives 74th Congress. Second session on H. J. Res. 449, 1936.

51. Gardner, L. U.: Pathology of acute silicosis. Am. Jour. Pub. Health, 23, 1240, 1933.

52. Sampson. H. L.: The roentgenograms in so-called acute silicosis. Am. Jour. Pub. Health, 23, 1237, 1933.

53. Ellman, P.: Pulmonary asbestosis, its clinical, radiological and pathological features and associated risk of tuberculous infection. Jour. Ind. Hyg., 15, 165, 1933.

53a. Teleky, L.: Der Berufliche Lungenkrebs Acta der Int. Vereinigung fur Krebsbekampfung, 3, 1938.

53b. Wedler, H. W.: Asbestosis and pulmonary carcinoma. Deut. Med. Woch. 69, 575, 1943.

53c. Siegal, William, Smith, A. R., and Greenburg, L.: The dust hazard in tremolite talc mining, including roentgenological findings in talc workers. Am. J. Roent. and Rad. Therap., 49, 11, 1943.

54. Lanza, A. J., McConnell, A. J. and Fehnel, J. W.: Effects of the Inhalation of asbestos dust on lungs of asbestos workers. Pub. Health Repts., 50, 1, 1935.

55. Commonwealth of Pennsylvania, Asbestosis. Spec. Bull., 42, 1935.

55a. Donnelly, J.: Pulmonary asbestosis, incidence and progress. Jour. Ind. Hyg., 1936.

55b. McPheeters, S. B.: A survey of a group of employees exposed to asbestos dust. Jour. Ind. Hyg. 18, 229, 1936.

56. Thompson, L. R., Brundage, D. K., Russell, A. E. and Bloomfield, J. J.: The health of workers in dusty trades. I. Health of workers in a Portland cement plant. Pub. Health Serv. Bull., 176, 1928.

57. Legge, R. T. and Rosencrantz, E.: Observations and studies on silicosis by diatomaceous silica. Am. Jour. Pub. Health, 22, 1055, 1932.

57a. McNally, Wm. D. and Trostler, I. S.: Severe pneumoconiosis caused by inhalation of fullers earth. J. Ind. Hyg. and Tox., 23, 118, 1941.

58. Commonwealth of Massachusetts, Report to the General Court of the Special Industrial Disease Commission. House No. 1350, 1934.

59. McConnell, W. J. and Fehnel, J. W.: Health hazards in the foundry industry. Jour. Ind. Hyg., 16, 227, 1934.

60. Warfield, L. M.: Results of the chest examinations of 2,500 in a heavy industry plant. Ind. Med., 4, 302, 1935.

60a. Kelly, J. F. and Hall, R. C.: Silicosis in modern foundries. N. Y. State Jour. Med., 37, 1, 1937.

60b. Osmond, L. H.: Dust hazard among foundrymen. Am. Jour. Roentgenol., 38, 122, 1937.

60c. Sander, O. A.: The lung findings in foundry workers. A four year survey. Paper read before Ind. Hyg. Section. A. P. H. A. at 66th Annual Meeting, N. Y. C., 1937.

60d. Trice, M. F. and Easom, H. F.: Report of a study of the foundry industry in North Carolina. N. C. State Bd. Health and N. C. Indust. Comm. Div. Indust. Hyg., Raleigh, 1938.

60e. Brown, E. W. and Klein, W. E.: Silicosis among naval foundrymen. U. S. Naval Med. Bull., 40, 42, 1942.

61. Hoffman, F. L.: The problem of dust phthisis in the granite-stone industry. U. S. Bur. Labor Stat., Bull. 293, 1922.

62. Russell, A. E., Britten, R. H., Thompson, L. R. and Bloomfield, J. J.: The health of workers in dusty trades. II. The exposure to siliceous dust (granite industry). Pub. Health Serv. Bull., 187, 1929.

63. Smith, A. R.: A study of granite cutting and granite cutters in the vicinity of New York City. Am. Jour. Pub. Health, 24, 821, 1934.

63a. Virginia State Dept. Health, Bur. Indust. Health: Silicosis in the monument manufacturing industry in Virginia. 1938.

64. Bloomfield, J. J. and Dreessen, W. C.: Silicosis among granite quarriers. Pub. Health Repts., 49, 679, 1934.

65. The health of workers in dusty trades. Exposure to the dusts of a silverware manufacturing plant. Pub. Health Serv. Bull., 28, 1933.

66. Smith, A. R.: Silicosis among metal polishers. Ind. Bull., 16, 6, 1937.

67. Higgins, E. and Lanza, A. J.: Pulmonary disease among miners in the Joplin District, Missouri, and its relation to rock dust in the mines. U. S. Bur. Mines Tech. Paper 10, 1915.

67a. Dreessen, W. C., Page, R. T. and Brenton, H. P.: Silicosis and other health problems of metal miners. Am. J. Pub. Health, 32, 142, 1942.

68. Childs, S. B. and Lanza, A. J.: Miners' consumption, a study of 433 cases of the disease among zinc miners in southwestern Missouri, with a chapter on roentgen ray findings in miners' consumption. Pub. Health Serv. Bull., 85, 1919.

69. Harrington, D. and Lanza, A. J.: Miners' consumption in the mines of Butte (Montana). U. S. Bur. Mines Tech. Paper 260, 1921.

70. Sayers, R. R., Meriwether, F. V., Lanza, A. J. and Adams, W. W.: Silicosis and tuberculosis among miners of the tri-state district of Oklahoma, Kansas and Missouri. U. S. Bur. Mines, Tech. Paper 545, 1933.

70a. Ellis, P. M., Smith, M. T., Bonebrake, H. E. and Hunter, L. B.: Silicosis: General considerations and survey in Coeur d'Alene mining district of Idaho. Northwest Med. 41, 406, 1942.

71. Sayers, R. R., Bloomfield, J. J., Dallavalle, J. M. et al.: Anthracosilicosis among hard coal miners. Pub. Health Serv. Bull. 221, 1935.

71a. Clarke, B. G. and Moffet, C. E.: Silicosis in soft coal miners. J. Ind. Hyg. and Tox., 23, 176, 1941.

72. Quaintance, P. A.: Silicosis, a study of 106 pottery workers. Am. Jour. Pub. Health, 24, 1244, 1934.

72a. Flinn, R. H., Dreessen, W. C., Edwards, T. I., Riley, E. C., Bloomfield, J. J., Sayers, R. R., Cadden, J. F. and Rothman, S. C.: Silicosis and Lead Poisoning among pottery workers. Public Health Bull. No. 244, 1939.

73. Smith, A. R. and Fehnel, J. W.: Silicosis among rock drillers, blasters and excavators in New York City based on a study of 208 examinations. Jour. Ind. Hyg., 11, 37, 1929.

74. Winslow, C. E. A., Greenburg, L. and Greenberg, D.: The dust hazard in the abrasive industry. Incidence of tuberculosis among workers exposed to mineral and metallic dusts. Pub. Health Repts., 34, 1171, 1919.

75. Greenburg, L. and Winslow, C. E. A.: The dust hazard in air-pressure abrasive blasting (sand blasting). Arch. f. Gewerbepath. u. Gewerbehyg. 3, 577, 1932.

76. Hayhurst, E. R. and Kindel, D. J.: Stereoscopic X-ray examination of sandstone quarry workers. Am. Jour. Pub. Health, 17, 818, 1927.

77. Dreessen, W. C.: Effects of certain silicate dusts on the lungs (talc and slate). Jour. Ind. Hyg., 15, 66, 1933.

78. National Safety Council. Final report of the spray coating committee, 1927.

79. Dreessen, W. C. and Dallavalle, J. M.: The effects of exposure to dust in two Georgia talc mills and mines. Pub. Health Repts., 50, 131, 1935.

80. Goldwater, L. J.: Incidence of silicosis in trap rock quarry workers as determined by X-ray. Jour. Ind. Hyg., 18, 550, 1936.

81. Hatch, T. and Pool, C. L.: Quantitation of impinger dust samples by dark field microscopy. Jour. Ind. Hyg., 16, 177, 1934.

82. Proceedings of Conference Concerning effects of Dust upon the Respiratory System. Industrial Commission of Wisconsin. Chicago, 1933.

83. Clark, W. I.: The dust hazard in the abrasive industry. Jour. Ind. Hyg., 11, 92, 1929.

84. Bloomfield, J. J. and Greenburg, L.: Sand and metallic abrasive blasting as an industrial health hazard. Jour. Ind. Hyg., 15, 184, 1933.

85. Greenburg, L., Siegal, W. and Smith, A. R.: Silicosis in the foundry industry. New York State Department of Labor, Spec. Bull. 197, 1938.

85a. Denny, J. J., Robson, W. D. and Irwin, D. A.: The prevention of silicosis by metallic aluminum. Canadian M. A. J. 40, 213, 1939.


THE ARMY MEDICAL LIBRARY, Washington, D.C.